THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—44TH YEAR SYDNEY, SATURDAY, DECEMBER 14, 1957 No. 24

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MODERN THOUGHT ON LESIONS OF THE SHOULDER REGION.[1]

BY M. NAOMI WING, M.B., B.S.,
Sydney.

MY interest in lesions of the shoulder region was aroused some years ago by the number of such patients whom I attended and the inadequacy of the treatment which was available. Out of 380 patients presenting at the Royal South Sydney Rheumatology Clinic, 33 complained of pain referred to the shoulder region. While overseas in 1953 visiting rheumatic clinics, I always inquired about the treatment they gave, and I was impressed with their outlook and results.

Voltaire has said: "What is true is not new, what is new is not true." When I have finished reading this paper, I hope to have refreshed your memory with what is true, and to leave you to try what is new, if you have not already done so, and to decide if it is true.

A search of the literature on this subject reminds us how slow has been the progress in our knowledge concerning the ætiology of this subject; but one important conclusion is that we have no more right to speak of a "frozen shoulder" as a final diagnosis without pin-pointing the affected underlying structures, than we have to speak of general peritonitis without naming the involved viscus in the abdomen.

It is only 122 years since the first mention of shoulder lesions was made in the literature, and this was by an English anatomist, J. G. Smith, in 1834. He described his dissections of 40 cadavera dead from varied causes, in seven of which there was gross disease of the shoulder joint; this he attributed to trauma. The conditions represented many of those which we frequently see today.

The term "periarthritis" was first mentioned by Duplay in 1872, and Baer-Painter, a radiographer, recognized calcified deposits in this region in 1907.

Until 1941 all the available references were from general or orthopædic surgeons; but since that time the physicians, especially those concerned with rheumatic disease, have been recognized as the authorities on this subject. It has become their misfortune to deal with these patients when their surgical colleagues have failed to relieve them.

In 1934 E. H. Codman, of Boston, published his book "The Shoulder". He is an orthopædic surgeon, but he gave a clear account of rupture of the rotator cuff, and also a detailed clinical picture of the syndrome which he referred to as "the frozen shoulder"; it has since then been called Codman's shoulder. We find him writing again in 1937. In *The American Heart Journal* in 1941, John M. Askey makes the first mention of a central cause in this condition in his article "The Syndrome of Painful Disability of the Shoulder and Hand, Complicating Coronary Occlusion".

[1] Read at the annual meeting of the Section of Physical Medicine and Rehabilitation (British Medical Association), Melbourne, November 13 and 14, 1956.

During World War II there was no literature on the subject; but in 1947 Otto Steinbrocker, of New York, wrote his first article, "The Shoulder Hand Syndrome, Associated Painful Homolateral Disability of the Shoulder and Hand with Swelling and Atrophy of the Hand". Mary Ropes and B. Frame, of Boston, also described this syndrome in 1948-1949. Since then

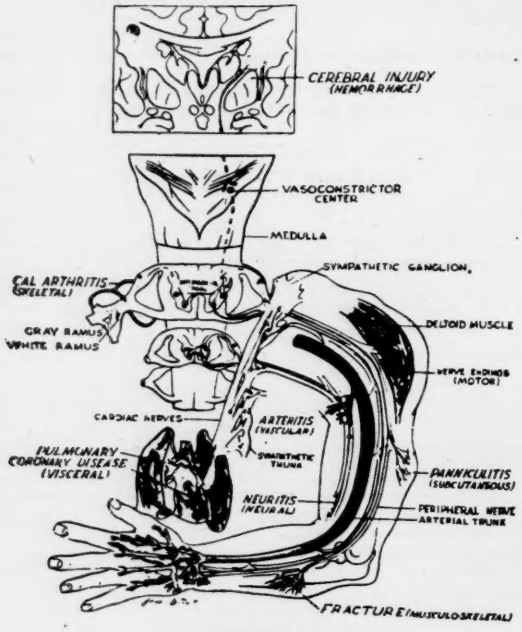

FIGURE I.

Mechanism of the shoulder-hand syndrome. (From Steinbrocker.)

Steinbrocker has evolved a plan of treatment based on his theory of the ætiology of this condition. We now find that two orthopædic surgeons, H. F. Moseley of Toronto, in his second edition of "Shoulder Lesions" (1954), and James Bateman, in "The Shoulder and its Environs" (1955), both refer to this theory as the accepted one.

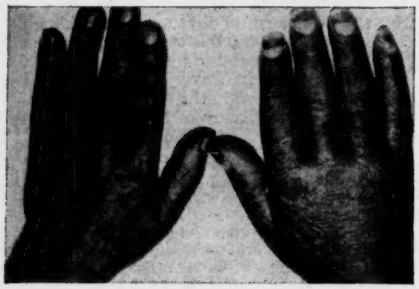

FIGURE II.

Post-herpetic shoulder-hand syndrome: affected hands with cyanotic swelling, desquamation of skin and trophic changes in nails. (From Steinbrocker.)

In the last-mentioned publication, Bateman has worked out his diagnosis on a clinical basis, using a detailed history of the pain for the purpose. He speaks of the following three types of pain: (i) shoulder-neck pain; (ii) lesions producing predominant shoulder pain; (iii) pain in the shoulder with radiating pain.

As in every other branch of medicine, a detailed history and careful physical examination are essential. In this area in

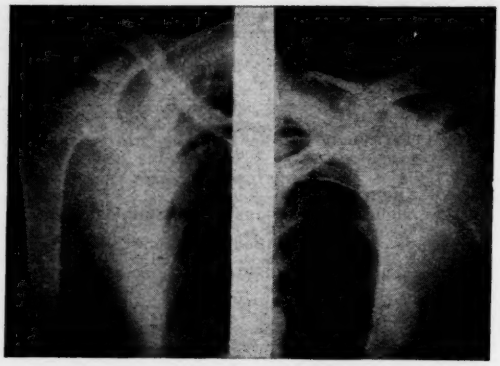

FIGURE III.

Post-herpetic shoulder-hand syndrome: osteoporosis of bones of shoulder. (From Steinbrocker.)

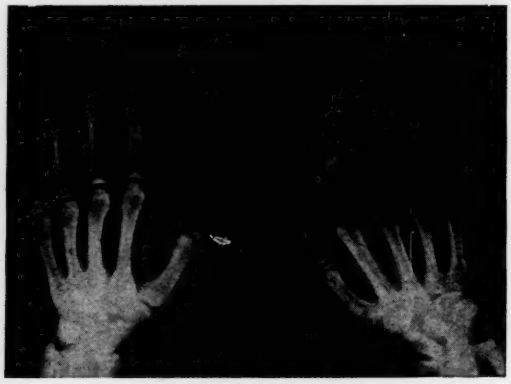

FIGURE IV.

Post-herpetic shoulder-hand syndrome: osteoporosis of hand and wrist. (From Steinbrocker.)

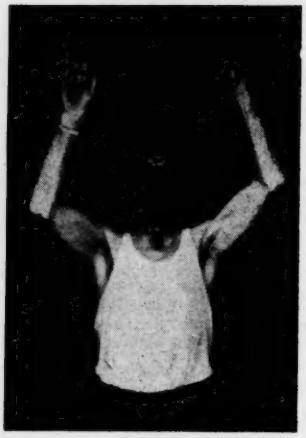

FIGURE V.

Bilateral shoulder-hand syndrome. Male, aged sixty-five years; myocardial infarction five months previously; bilateral limitation of abduction. (From Steinbrocker.)

particular, where many of the causes are central as well as local, it is important to have both a general and a local history, and to make a general physical examination. The history of the pain should include its relation to posture, occupation, injury and movement, and also the character of the pain and its ramifications; for example it may radiate to the outer or inner borders of the hand, or the whole hand may be involved in lesions of the neuro-vascular bundle.

FIGURE VI.

Same patient as in Figure V: limitation of internal rotation. (From Steinbrocker.)

DIFFERENTIAL DIAGNOSIS OF SHOULDER-NECK PAIN.

The differential diagnosis of shoulder-neck pain may be set out in tabular form as follows:

- Postural disturbances.
- Fibrositis.
- Muscle, fascial and ligamentous injuries.
- Subluxation of cervical facets.
- *Ligamentum nuchæ* injuries and whiplash syndrome.
- Arthritis of the cervical part of the spine.
- Scapulo-thoracic lesions.
- Neurological disturbances:
  - Trapezius paralysis.
  - *Serratus anterior* paralysis.
  - Deltoid paralysis.
  - Progressive muscular atrophy and allied lesions.
- True neck disorders:
  - Anterior neck pain:
    - Acute adenitis.
    - Infection of the pharynx.
    - Congenital abnormalities.
  - Posterior neck pain
  - Meningitis.
  - Uncommon conditions.
- Referred pain:
  - Pulmonary sulcus lesions.
  - Cardiac disease.

This type of pain arises from disease in the posterior scapular, interscapular and adjacent neck areas. It is mediated through branches of the posterior rami of spinal nerves. This pain has an aching, gnawing quality that seems to encompass the whole shoulder, and localization is difficult. This area contains mostly muscle and fibrous tissue, and the complaints arise out of everyday activities, and are not associated with specific muscle action or particular injury.

DIFFERENTIAL DIAGNOSIS OF LESIONS PRODUCING PREDOMINANT SHOULDER PAIN.

The lesions producing predominant shoulder pain may be tabulated as follows:

- Degenerative tendinitis of rotator cuff.
- Tendinitis with calcification.
- Tears of rotator cuff.
- Bicipital lesions.
- Acromio-clavicular arthritis.
- Adhesive capsulitis.
- Neoplasms.
- Less common conditions:
  - Osteoarthritis.
  - Infective arthritis.
  - Metabolic arthritis.
  - Neurological lesions.

The pain may be referred from the neck, breast, heart, gall-bladder or gastro-intestinal tract. These lesions are recorded through branches of the brachial plexus to the joint. They arise from disease of the gleno-humeral joint proper, more often than from any other cause. The cardinal symptoms are clearly localized pain and soreness, the patient grasping the point of the shoulder with the hand of the good side. Movement of the shoulder influences the pain more than any other action. Most of the acute shoulder disabilities belong in this group, and progressive limitation of movement accompanies the pain.

FIGURE VII.

Puffiness of digits, especially on the left, and œdema of dorsa of hands. (From Steinbrocker.)

DIFFERENTIAL DIAGNOSIS OF SHOULDER AND RADIATING PAIN.

The differential diagnosis of shoulder and radiating pain may be set out as follows:

- Cervical root syndrome:
  - Extruded intervertebral disk.
  - Intraforaminal root compression.
  - Cord tumours.
- Cervical rib and scalene syndromes.
- Clavi-pectoral compression syndromes:
  - Costo-clavicular syndromes.
  - Postural syndromes.
  - Sleep syndromes.
  - Hyperabduction syndromes.
- Reflex dystrophy of the upper extremity (shoulder-hand syndrome).

FIGURE VIII.

Same patient as in Figure V: effort to make fist; stiffness and limitation of interphalangeal movements. (From Steinbrocker.)

This group of disorders produces some shoulder discomfort; but pain in the forearm or hand becomes the most unpleasant symptom, and persists as the most disturbing element. Two fundamental underlying causes may be recognized, those with a dominant neural background, and those with a vascular and a neuro-vascular association. In the latter, the pain is a dull, burning ache, rather than the lancinating, sharp pain of nerve root irritation. As the last of these conditions is of particular interest to the physician, I propose to deal with it separately, and to show you some slides which were given to me by Dr. O. Steinbrocker.

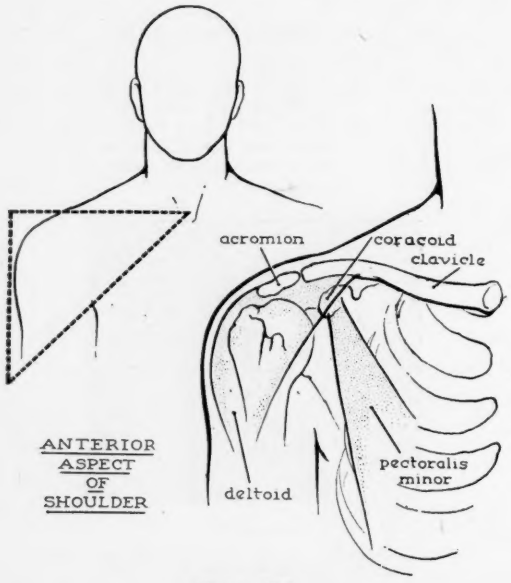

FIGURE IX.

Anatomy of anterior aspect of shoulder: anterior triangle. (Bateman, 1955.)

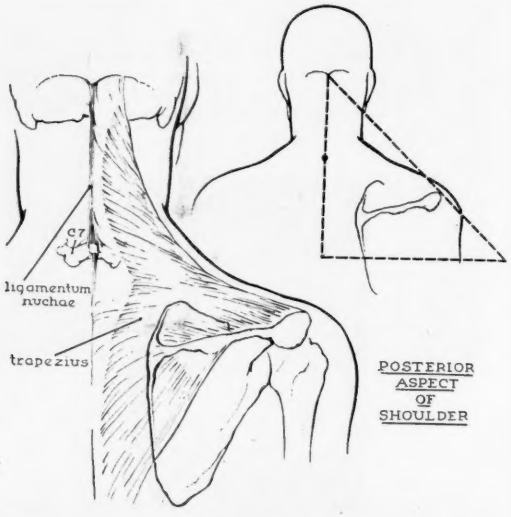

FIGURE XI.

Posterior aspect of shoulder: posterior triangle. (Bateman, 1955.)

THE SHOULDER-HAND SYNDROME.

Dr. Steinbrocker has defined shoulder-hand syndrome as "a painful disability of the shoulder and hand of the same extremity, with swelling and vasomotor features of the hand and fingers where progression to trophic changes and contractures may occur". This syndrome may present at any stage, either with a swollen hand alone or concurrently with shoulder involvement; or the shoulder alone may suffer. Nevertheless, the underlying mechanism is the same. Figure I offers a simple working explanation of the mechanism. It appears to be a reflex motor and neuro-vascular reaction starting from an area of local tissue injury or irritation, such as a traumatized extremity, a myocardial infarction, a gastric hæmorrhage from peptic ulcer or some other provocative focus. Impulses from such a site of excitation travel centrally along the afferent nerve roots to enter the internuncial system of neurons within the spinal cord. Some of these neurons intercommunicate with each other and excite one another. In this way continuous activity is maintained in the cord for prolonged periods at the anatomical level related to the lesion. This stimulative activity is discharged through interconnecting neurons in the lateral autonomic horn to provoke autonomic or sympathetic effects, and in the anterior horn of the cord to produce motor reactions (muscular contraction) through their outgoing peripheral pathways. This incessant stimulation is expressed peripherally by motor and neurovascular symptoms which make up the features of reflex

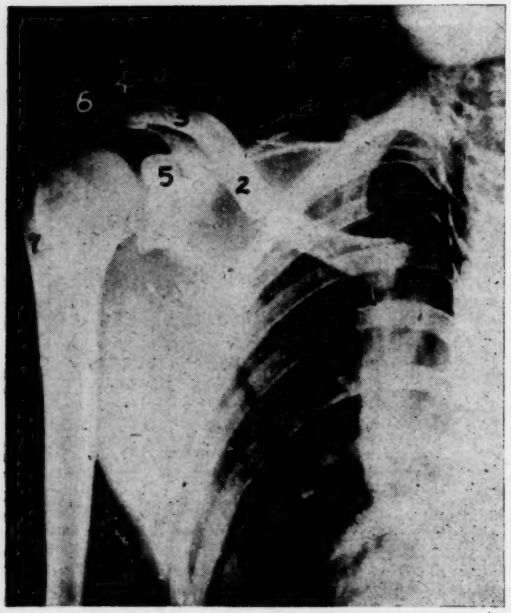

FIGURE X.

Skiagram of anterior aspect of shoulder: 1, sternoclavicular joint; 2, junction of clavicle curves; 3, suspensory ligament attachment; 4, acromio-clavicular joint; 5, coracoid process; 6, acromion; 7, bicipital groove. (Bateman, 1955.)

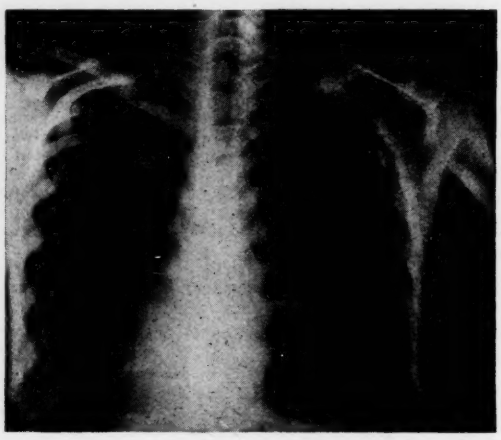

FIGURE XII.

Posterior anatomy of shoulder: 1, body of scapula with projecting medial angle; 2, clavicle from posterior aspect; 3, thoracic cage overlapped by scapula. (Bateman, 1955.)

dystrophy. An understanding of these principles will prevent many of the mistakes in treatment, which we so frequently see.

Figures II, III and IV demonstrate the early signs and symptoms.

In the hand the patient complains of pain with or without cutaneous hyperæsthesia, vasomotor disturbances including changes in skin temperature, and limited movement. Swelling is present with atrophic changes in the integument, and ground glass or spotty osteoporosis is apparent in the X-ray films.

In the shoulder the pain is continuous, and there is severe spasm in all muscles; they are swollen and tender, and localized masses may even be present. Loss of movement is pronounced, leading to atrophy of all muscles of the shoulder girdle with osteoporosis of the upper end of the humerus.

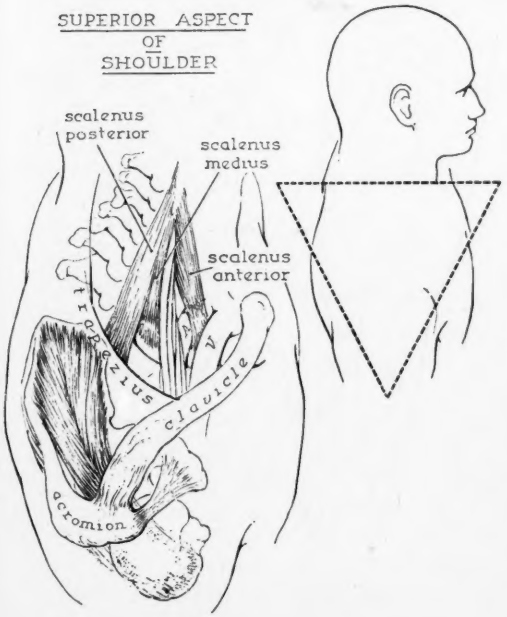

FIGURE XIII.
Anatomy of superior aspect of shoulder: superior triangle. (Bateman, 1955.)

In Figures II, III and IV the condition is post-herpetic; in Figures V, VI, VII and VIII the condition followed myocardial infarction.

Dr. Steinbrocker states that there are three phases of this syndrome, as follows. The first phase, demonstrated in Figures II, III and IV, lasts for three to six months. The second phase, as demonstrated in Figures V, VI, VII and VIII, lasts for three to six months. The third phase is a period of trophic change, ranging from recovery to irreversible changes in shoulder and hand. These may be muscular weakness, gradual atrophy and contraction of the fingers and residual disability of the shoulder. Sometimes there is a rapid progression to these alterations from the onset of the syndrome.

The length of time necessary to recover depends largely on correct early diagnosis and prophylactic care, and an understanding of the underlying mechanism, so that further trauma will not be inflicted by the treatment.

The changes in the hand have been described by Sudek. They are apparently the result of persistent pain and irritation, which produce a preliminary sympathetic paralysis; this would account for the vasodilatation seen clinically. This is later followed by sympathetic over-activity resulting in vasoconstriction. The later changes are due to vasoconstriction, in the following way. The early changes in the hand are caused by serum leaking through damaged capillaries into the surrounding tissues and permeating the capsules and ligaments of small joints. Unless some effort is made to disperse this transudate, which is rich in fibrin, it becomes deposited as scar tissue. As elsewhere, as this scar tissue matures it contracts and renders immobile those tissues which it permeates. If treatment is delayed for more than three months, the scar tissue is so well established that nothing will make any difference to it. The osteoporosis seen in the X-ray film of the hand is due to the hyperæmia in the early stages.

Treatment.

The way of treatment is long and tedious in these cases, but the end result can be very rewarding. If you ever have the misfortune to experience this painful disability yourself, then, and only then, will you realize the demoralizing effect of the continuous pain, loss of sleep and loss of function.

When speaking to a group such as this, one does not need to emphasize the fact that the shoulder-hand syndrome is the result, not the cause, and also that a positive diagnosis of the cause must be established before a programme of treatment is instituted. Any one of the conditions named in the differential diagnosis can lead to sympathetic irritation, if restriction of

FIGURE XIV.
Skiagram of superior aspect of shoulder: 1, coracoid process; 2, acromion process; 3, lesser tuberosity; 4, greater tuberosity; 5, glenoid cavity; 6, acromio-clavicular joint. (Bateman, 1955.)

movement of the shoulder is prolonged, causing continued irritation in the nervous system. The numerous general conditions that may cause the syndrome demand special investigations according to their symptomatology. I regard as a minimum a full blood count and estimation of the erythrocyte sedimentation rate, with an X-ray examination of the shoulder, neck and hand. To this should be added X-ray examination with a barium meal, electrocardiography *etc.*, as required. Treatment resolves itself into (i) treatment of the underlying cause, (ii) treatment of the sympathetic upset and (iii) treatment of the affected structures of the shoulder and hand.

Treatment of the Underlying Cause.

Treatment of the underlying cause should be thorough, and the patient should be kept under observation, because recurrence

of the condition may cause exacerbation of the shoulder symptoms already existing, or may even cause them to appear in the unaffected limb.

This happened to one of my patients, who had a severe exacerbation of her peptic ulcer in November, 1955, resulting in left shoulder-hand syndrome. This was responding to treatment very well. She went away for the Christmas holidays, was indiscreet in her diet, and suffered an exacerbation of her ulcer. She returned at the end of January with the syndrome established in the right shoulder. The left arm is now completely recovered after one year, and the right arm is beginning to recover. Incidentally, she had received attention to the right shoulder, in the form of passive movements and gentle exercises; but it did not seem to prevent the condition.

Treatment of the Sympathetic Upset.

Steinbrocker has evolved a method of treatment by continuous drip administration of procaine solution down to the stellate ganglion through a polythene catheter. This necessitates the patient's admission to hospital and constant supervision; but the patients whom he showed me had considerable relief of pain after forty-eight hours and were very grateful. This treatment is continued for ten days.

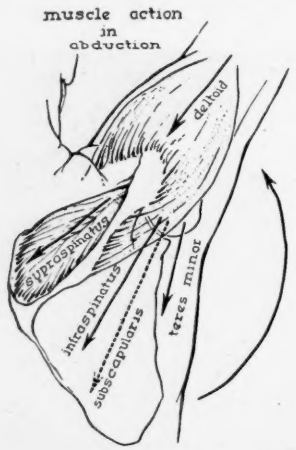

FIGURE XV.
Abduction mechanism. (Bateman, 1955.)

Intermittent ganglion blocking is very effective in skilled hands; but it has complications, and should not be undertaken in a consulting room or out-patient department unless suitable measures are ready to deal with these complications.

In one of our cases, in which this was performed by a most competent neurosurgeon, there was a projection of the spinal theca, and he had the misfortune, in spite of aspiration, to inject "Xylocaine" into the sac. The patient immediately stopped breathing, and it was six hours before she was safe to be left.

Hydergine is recommended as a general sedative of the autonomic nervous system, and it does seem to help in some cases. I have made it my practice to elicit a history of migraine, as indicating an underlying instability of the autonomic nervous system. It is surprising how often it is present.

Reconditioning of the Affected Structures.

I have certain "Do's" and "Don'ts" about reconditioning of the affected structures.

The "don'ts" are as follows: (i) Don't manipulate shoulders with this condition. (ii) Don't put them up in abduction splints. The latter procedure encourages contracted muscles to shorten while in their shortest position of the splint. The former adds irritation to the already over-excited nervous system, and does harm. Some of my most difficult tasks have been to undo harm thus done. In support of these statements I quote H. F. Mosely (1953). He has obtained the personal opinion of Lorenz Böhler, whom the orthopædic surgeons quote as the authority for the use of the abduction frame in lesions of all kinds in this area.

Since 1935 I have only exceptionally used the abduction frame for fractures of the humerus, and since that year, I have mentioned this fact in my book. I therefore consider that the abduction frame should only be used on rare occasions.

This concerns traumatic lesions, but applies to all lesions of the shoulder region.

The "do's" are as follows. (i) Adopt conservative measures to relieve pain and promote sleep by giving tranquillizing agents—for example, "Nutinal" or "Nembudeine" for sleep, and salicylates or "Butazolidin" for pain. If the last-mentioned is not effective after forty-eight hours, discontinue its use; its side effects can be harmful to the underlying cause. Do not use drugs of addiction. (ii) Institute prophylactic care of the affected hand and shoulder, to preserve function and prevent

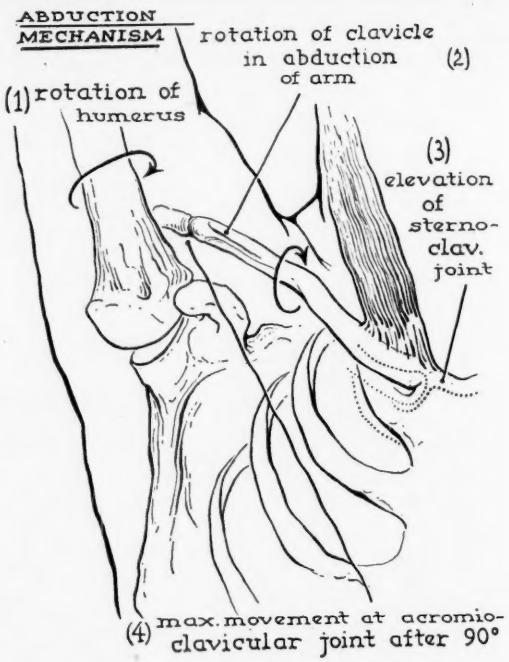
ABDUCTION MECHANISM
(1) rotation of humerus
rotation of clavicle in abduction of arm (2)
(3) elevation of sterno-clav. joint
(4) max. movement at acromio-clavicular joint after 90°

FIGURE XVI.
Abduction mechanism. (Bateman, 1955.)

deformity. For this purpose I use the following method: (*a*) Hydrocortisone and procaine injections are given into the structures of the shoulder region—that is, the shoulder joint itself, the tendon of the long head of the biceps, the supraspinatus and infraspinatus tendons, the underlying bursæ, and the fascia along the vertebral border of the scapula. These injections relieve spasm and inflammatory œdema, and may be repeated weekly. (*b*) Heat is applied, in the form of infra-red, short-wave or ultrasonic therapy; any one of these alone is used to promote relaxation, but ultrasonic therapy must not be combined with other forms of heat treatment. (*c*) Massage is carried out. This should be light stroking in the early stages, for relaxation; as pain and tenderness disappear it can be changed to deep kneading massage, to recondition the muscles. (*d*) Exercises and movement are applied to all joints. A full range of passive movement is carried out daily, to the point of pain, in shoulder, elbow, wirst and fingers. Assisted active exercises with pulleys may be undertaken as pain diminishes, and also gravity-assisted exercises. (*e*) The hand needs special care. Ultrasonic therapy under water is given daily, followed by wax baths with the hand elevated. This may be done in a cage with warm air blown in. Exercises for the intrinsic muscles are carried out while the hand is in the wax. These should not last longer than three minutes, to avoid fatigue. Circumduction of the arm should be the aim of all treatment, with full function of the hand. This is possible in every case, if treatment is instituted early. Before ordering special movements, one should remember the purpose of the shoulder girdle, which is to suspend the arm, to stabilize its movements and to mobilize the arm and hand. (See Figures IX, X, XI, XII, XIII and XIV.)

Suspension is achieved by a combination of the strut of the clavicle and its strong attachment to the scapula. Stability is

attained by the scapula and contraction of the flat muscles attached to its vertebral border—namely, the trapezius and the *serratus anterior*. Mobility is effected by contraction of the deltoid muscles of the rotator cuff and the pectorals. To circumduct the arm (Figures XV and XVI), the scapula is fixed to the thorax by the flat muscles. The head of the humerus is snubbed into the glenoid cavity by the rotators, while the deltoid contracts simultaneously. Before the arm can be abducted beyond 90°, the greater tuberosity of the humerus to which these structures are attached has to pass under the strong coraco-acromial arch. This it does by external rotation of the arm by contraction of the infraspinatus, the *teres minor* and the posterior deltoid.

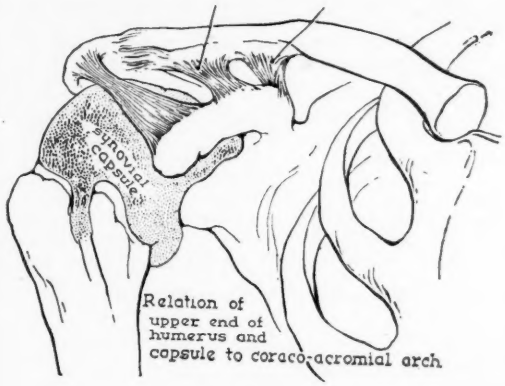

FIGURE XVII.
Coraco-acromial arch. (Bateman, 1955.)

All exercises are, therefore, designed to restore external rotation first and then the other movements in turn. The gravity-assisted exercise with arms hanging is designed to gain balanced relaxation of gleno-humeral movement, and increased range of movement in the shoulder girdle to promote stabilizing action of the rotators.

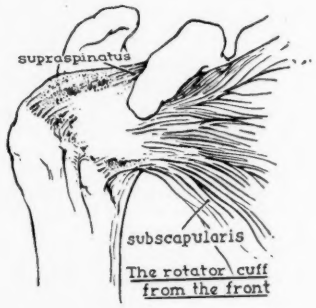

FIGURE XVIII.
Capsule from the front.
(Bateman, 1955.)

Occupational therapy should be combined with physiotherapy in the programme of treatment, preferably in a rehabilitation centre, where the whole team works with the patient.

SUMMARY.

Regard every patient with pain in the shoulder as presenting a serious clinical problem. Search until you find the underlying cause. Treat that as well as the shoulder and hand involved. Be conservative but concise, and have a positive outlook to give your patient confidence and moral support. Organize your programme of treatment, and acquaint yourself with the mechanism of the condition and the anatomy of the structures involved. Supervise these patients, continually changing the physiotherapy whenever pain is provoked. Then, and only then, will you achieve full function of the shoulder and hand, which should be your goal.

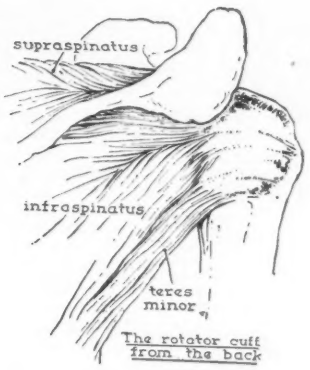

FIGURE XIX.
The capsule from the back. (Bateman, 1955.)

ACKNOWLEDGEMENTS.

I am grateful to Dr. O. Steinbrocker for having given me a number of slides; from these Figures I to VIII are taken. Dr. J. E. Bateman and the C. V. Mosby Company have given me permission to reproduce the remaining 11 illustrations from his book (*Bateman, 1955*).

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LOW BACK PAIN AND ALLIED SYMPTOMS, AND TREATMENT RESULTS.[1]

By G. D. MAITLAND, M.A.P.A.,
Adelaide.

IN recent times there has been a considerable amount of discussion on disk lesions and manipulation. No attempt will be made to enter into the field of diagnosis of these conditions, as this is not within the province of physiotherapy; but it is the aim of this article to bring to notice a summary of the results of a survey which was made in South Australia during 1956 by G. D. Maitland and M. J. Paige under the auspices of the University of Adelaide General Research Committee.

The survey was carried out in three separate theatres—a country centre, a city practice and a public hospital—

[1] Part of this paper was presented at a post-graduate lecture to members of the Australian Physiotherapy Association in South Australia, June, 1957.

and was the first year of a three-year project which concerns only lumbar pain and pain referred from disorders in the lumbar part of the spine in patients who have been sent for physiotherapy.

The standard required for the relief of symptoms was gauged by the number of treatments necessary for the patient to resume normal function. The manipulations used were all manual and without sedatives or anæsthesia, changes being gauged by testing the patient's range of movement and/or degree of pain, between all manœuvres.

There were 342 patients, of whom 122 were eliminated because they fell under one of the following headings: (*a*) Patients with a very low pain threshold or with excessive functional overlay (24 patients). (*b*) Patients (36) whose relief of pain could have been classified as spontaneous recovery (for classification all patients were required to have been *in statu quo* or to have been deteriorating before treatment started). This group also includes another 36 people who just stopped treatment or whose charts were inaccurate. (*c*) Patients (26) with other pains, such as pain on hip movement or sacro-iliac joint movement, *etc.* This left a total of 220 for the survey, of whom two-thirds were male and one-third female.

It was found that patients whose pain took less than three days to reach its final area or full intensity responded better than those taking three days or longer. Also, the presence of neurological changes influenced the result unfavourably. This more acute group without neurological changes will be considered in the first part, which is by far the larger part. The second part deals with "gradual onset" and patients with neurological changes. Other factors also influencing prognosis were (i) degree of radiation of pain downwards, and (ii) the presence or absence of deformity (sciatic scoliosis or lumbar kyphosis). Therefore the first part is subgrouped as follows: Group A: Pain in the lower back region only (51%); Group B: pain in the back and/or the buttock region (14%); Group C: pain from the back extending down as far as the knee, but not beyond (19%); Group D: pain from the back down to the lower part of the leg or the foot (16%). In each of these groups the influence of the presence of deformity will be considered.

Section I: Acute Onset.

Group A.

As this group had the largest number of patients, it is the most accurate in its percentages. It is interesting also, in that the patients were treated by two different methods (Figure I).

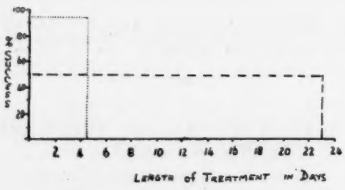


FIGURE I.

Comparative treatment results for back pain only; interrupted line, heat and exercises; dotted line, manipulation.

One section of 20 patients received treatment by heat together with back and postural exercises. This gave a 50% success, which took an average of 23 days' treatment each. Of the 10 "failures", six were then treated by manipulative methods, and all were relieved in an average of six days' treatment.

The other section, consisting of the six patients from the "failures" above and 75 others, making 81 in all, received treatment by manipulation. There was a 96% success, which took an average of 4·5 treatments each (Figure II). Those with gross deformity in this group (*i.e.*, the sciatic type scoliosis) were relieved in 91% of cases in an average of six treatments, so it can be seen that the presence of deformity did not make much difference here (Figure III).

Group B.

In this group of patients, with buttock pain, there was a 95% success with manipulative treatments in an average of four treatments (Figure II). Deformity in this group, as previously, did not make any material difference to the result (Figure III).

Group C.

The result in this group of patients with pain in the upper part of the leg and back was 96% in an average of 5·7 manipulative treatments. This indicates that as the pain extends further from the spine, so it takes longer to relieve, but is relieved in approximately the same percentage of cases (Figure II). The presence of deformity in this group reduced the percentage successful result to 60, and these took an average of 11 manipulative treatments (Figure III).

Group D.

The percentage of successes dropped slightly and the number of treatments increased, in this group of patients with pain in the lower part of the leg and/or the foot. There was 91% success, which took an average of seven manipulative treatments to achieve (Figure II). Deformity

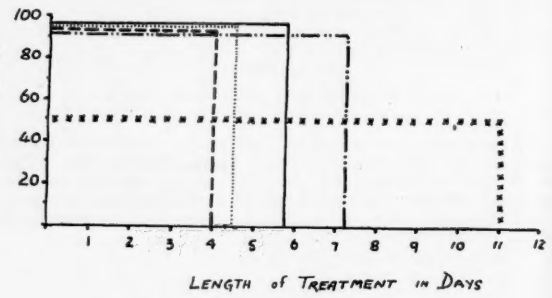


FIGURE II.

Manipulative treatment results; dotted line, Group A—back pain only; interrupted line, Group B—back pain and buttock pain; solid line, Group C—back and thigh pain; dashes and dots, Group D—back to foot pain; crosses, gradual onset and neurological sign group.

again made a big difference and caused the success percentage to drop to 50 in an average of nine treatments by manipulation.

Summary.

The results in these four groups may be summarized as follows: (i) Signs such as tenderness or limitation of movement did not materially affect the result of treatment by manipulation or the types of manipulations used. (ii) Neither sex nor age had any material bearing on the final result of treatment. (iii) The duration of the pain between its onset and the commencement of treatment was associated with only a slight variation in the result of treatment. Shorter duration of pain yielded a greater percentage success. (iv) Deformity made little or no difference in the groups with back pain only or back and buttock pain; but in the group with pain in the upper part of the leg, the result dropped from 96% to 60% success, and in the groups with pain in the lower part of the leg and the foot the result dropped from 91% to 50% success (Figure III).

Section II: Gradual Onset and Neurological Signs.

Let us return now to the much smaller second section mentioned earlier in this article. This includes the

following patients: (*a*) patients whose pain took three days or longer to reach full intensity or its final area (*i.e.*, very gradual onset); (*b*) patients who had neurological signs, such as changes in sensation or reflexes, muscle weaknesses or wasting, together with dermatome areas of pain. There is considerable overlapping here, as the pain of 34% of the patients with neurological changes had a very gradual onset. As the numbers were small in this part, the results can only be classed as a guide. In the cases of very gradual onset, only a 50% success was achieved with an average of 11 treatments. When neurological changes were present, in 54% of cases a response was achieved with an average of nine treatments.

Conclusion.

Not one patient was made worse to a harmful degree whether there were any deformities or neurological signs present or not, and the percentage of success was 50 or 90, depending on type; this was achieved in four to 11 treatments, usually daily.

These facts must show that manipulation, as a form of treatment for patients with low back pain and referred symptoms, must be considered favourably, since a definite result indicating its effective use in any particular case is reached within a fortnight.

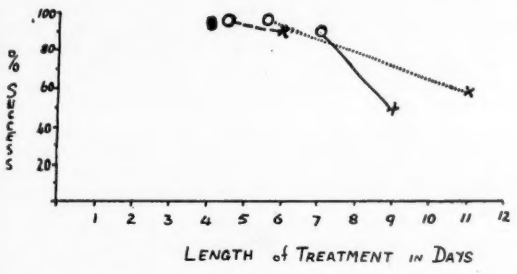


FIGURE III.

The effect of deformity on treatment results; open circles, without deformity; crosses, with deformity; interrupted line, Group A—back pain only; solid circle, Group B—back and buttock pain; dotted line, Group C—back and thigh pain; solid line, Group D—back to foot pain.

Summary.

In over 90% of all patients with a sudden "two-day onset" of pain, no neurological changes and no deformity, relief of their symptoms was obtained with an average of six manipulations, the results being best when the pain was localized to the back and deteriorating if the radiation extended down the leg. The presence of deformity—that is, sciatic scoliosis—necessitated more manipulations and gave a poorer prognosis, especially for patients with pain down the leg.

Of patients with pain of very gradual onset or with neurological signs, only half were relieved, and an average of 11 manipulations was required.

Acknowledgements.

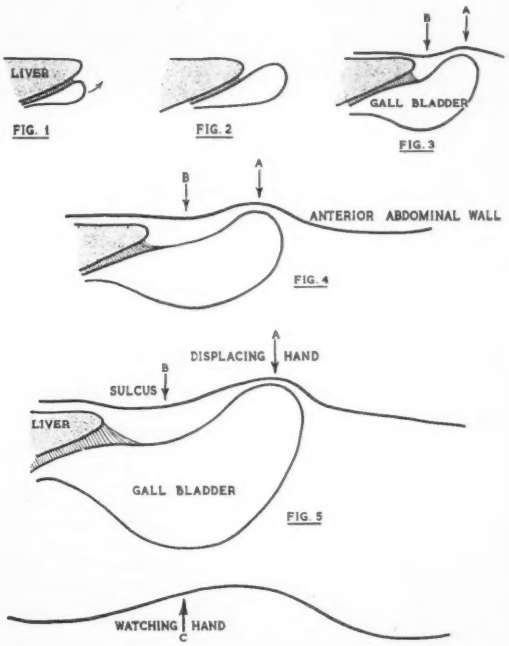
I should like to express my thanks for the help and encouragement I have received from Mr. W. J. Betts, M.B., B.S., (Adelaide), M.Ch.Orth. (Liverpool), F.R.A.C.S., and from Mr. Lansell Bonnin, M.Ch.Orth., F.R.C.S., both of Adelaide. They have been of considerable help both in the presentation of the results and in the preparation of the paper. As the majority of the patients used for this survey were referred for treatment by them, it is clear that they were in the most favourable position to check the paper and verify the results. I have been very happy to have their support.

ENLARGEMENT OF THE GALL-BLADDER AS A CLINICAL SIGN.

By P. A. TOMLINSON,
Sydney.

THE normal gall-bladder may be said to have one aspect covered by peritoneum, and to be attached by the other aspect to the liver and the lesser omentum. This attachment is by areolar tissue, fibrous tissue, blood-vessels and the cystic duct.

Not infrequently the gall-bladder has a mesentery of varying degrees of completeness. This mesentery too serves for attachment to or suspension from the liver. Notwithstanding this variability of attachment to the liver, the postero-inferior aspect of the gall-bladder is almost always free to expand, whereas it is more difficult for the antero-superior aspect to become elongated, owing to its attachment to the liver. The gall-bladder therefore, when it becomes enlarged, does not distend uniformly and spherically, but becomes more retort-shaped in outline. The effects of this method of enlargement are as follows: (i) The abdominal wall, especially as seen in a tangential view, may be noticeably pushed forward near the lowest portion of the palpable mass, not at the middle of the "tumour". (ii) There is often a distinct sulcus between this prominence of the gall-bladder and the liver edge. (iii) The mass is easily defined on palpation, and more elongated in the vertical than in the transverse direction. (iv) It is movable from side to side, but not to any great extent from above downwards. (v) On occasion it is large enough to be ballotted bimanually between the hand on the anterior abdominal wall and the hand in the right loin, and can be felt to return to the abdominal wall after displacement. This bimanual palpation of the greatly enlarged gall-bladder is best carried out with the posterior hand at a slightly higher level than the anterior hand. The anterior (displacing) hand is directly applied over the visible tumour; the posterior (watching) hand is opposite the groove between the gall-bladder fundus and the liver edge. (vi) The tumour mass moves up and down on respiration. (vii) Respiration is free, unimpeded and painless.



The foregoing signs are to be found in the progressive enlargement of the gall-bladder in a patient with an obstruction of the cystic duct (usually by stone, the patient not being jaundiced), an obstruction at the junction of the cystic and common hepatic ducts (the patient jaundiced—a rare exception to Courvoisier's law), or a common bile duct obstruction from reasons other than stone (usually by neoplasm, occasionally by inflammation of the pancreas, ampulla or common bile duct itself, occasionally from pressure from without). In common bile duct obstruction by stone, a palpable gall-bladder is uncommon, owing to previous gall-bladder inflammation and fibrosis.

Though in these patients with gall-bladder enlargement associated with and resulting from duct obstruction the palpable mass may be tender, there is an absence of the signs of peritoneal inflammation, and the outline of the mass is readily defined (*i.e.*, there are seldom adhesions).

In the enlarged palpable gall-bladder of acute cholecystitis, with or without stone, the organ is not enlarged to such a size as to be palpable bimanually, tenderness is exquisite, and there are signs of local peritonitis (hyperæsthesia and rigidity). These, together with the fact that omental and other adhesions are present, result in the enlargement's being less easily definable and frequently immobile. Deep breathing causes pain, with the result that respiration is often shallow and impeded.

THE PSYCHOLOGICAL SEQUELÆ TO MURRAY VALLEY ENCEPHALITIS.

By A. W. MEADOWS, M.A., Ph.D., F.B.Ps.S., A.I.S.,
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MURRAY VALLEY ENCEPHALITIS (1951) is an epidemic neurotropic disease reported by several investigators (Anderson, 1952; Anderson *et alii*, 1952; French, 1952; Robertson, 1952; Robertson and McLorinan, 1952; Burnet, 1952). It was identified as being similar to the Australian "X" disease reported by Cleland, Campbell and Bradley (1917) and also related to the Japanese "B" type (Olitsky and Casals, in Rivers, 1948). The mortality in Murray Valley encephalitis (45%) was not as high as Australian "X" disease (70%), but was in keeping with European estimates made in 1921 and 1922, which ranged from 27% to 50%. There were 40 recognized cases of Murray Valley encephalitis with 23 survivors, compared with 134 cases of Australian "X" disease with 40 survivors. In the Australian "X" disease, after one year it was found that of the 40 survivors, 35 had recovered completely, *i.e.*, showed no disability—three had subsequent paralysis, and two showed signs of mental disorder. No further follow-up studies on these patients had been carried out, and there was no information as to the completeness of the recovery.

However, in Europe, the sequelæ to attacks of the disease have suggested an unfavourable prognosis for 50% of patients who recover (Walshe, 1946). It has also generally been suggested that during convalescence or up to three years later the Parkinsonian syndrome developed, with an extra-pyramidal type of muscular rigidity, slow restricted movement and tremor spreading from limb to limb. It is of importance to note also that the earliest reported signs to be significantly connected with the encephalitis were of physical origin.

It is the purpose of this communication to suggest that, prior to the onset of the Parkinsonian syndrome, there is an earlier stage of organic deterioration which manifests itself mainly in the psychological condition of the patient. This early deterioration was not previously detected, because the deviation from normal conduct and behaviour could be disclosed only by subjecting the patient to rigorous psychological examination. It is true that some hint of its existence could have been gained from vague complaints of patients that they had "wandering pains" or memory failures, slight aphasia or blocking of thought processes. But it was generally thought that the complaints of encephalitis patients were disproportionate to their apparently healthy state and to what was revealed by medical examination. Not infrequently, therefore, the symptoms were accepted as manifestations of an hysterical or generally neurotic character. With children the early symptoms of refractoriness, delinquency and wandering were attributed to the encephalitis, and Walshe (1946) goes so far as to state that mental and moral deterioration sometimes follow on encephalitis in children.

In the light of this knowledge, it was decided to make a psychological examination and provide if possible a systematic description of the psychological state following the Murray Valley encephalitis epidemic of 1951. The survey, made in 1952 and again in 1953, of all undoubted cases was completed under the direction of the Victorian Mental Hygiene Department, in cooperation with the Department of Health, the Walter and Eliza Hall Institute of Medical Research, and the local health officers and general practitioners in Victoria.

Examination.

The examination time for each patient was approximately two to three hours, and *rapport* was successfully established in all cases, only one showing preliminary difficulty. In general the psychological examination was preceded by a brief neurological examination. Histories were compiled from practitioners' records and interviews with parents and relatives in order to obtain: (i) a picture of the patient's behaviour, habits, abilities, moods, *etc.*, before the disease; (ii) the behaviour during the incubation period and illness; (iii) the behaviour during the first three months of convalescence; (iv) any changes occurring at the intervals of six, 12 and 18 months after the illness.

The following tests were used in the psychological examination of patients: for adults, Wechsler-Bellevue Form I, Cornell-Index N and 16 P.F. test; for children, children's Wechsler, Terman-Merrill Form L, Vineland Social Maturity, Bender, Rosenzweig's Picture Frustration, Behn test and Burt's Reading Scale. The object was to discover (i) the present intellectual performance, (ii) the past intellectual performance, (iii) the presence of mental deficit, and whether this was attributable to Murray Valley encephalitis; (iv) any overt psychoneurotic signs, (v) general personality reactions to a severe illness, (vi) any psychological concomitants of organic involvement suggesting Parkinsonism.

Results.

Age.

The 20 patients examined comprised seven adults and 13 children. The age distribution of the patients is given in Table I.

Neurological Weakness.

The neurological examination revealed certain muscular weaknesses, in four cases right-sided (arm, leg, shoulder or both). In only one case was left-sided weakness present, again not severe. In one case there were tremors, with some jerky movements, and the handwriting of this patient was impaired. The weaknesses may be divided into severe and less severe muscular weakness, as follows:

Serious weakness with wasting and restricted movement, continuing in arms, shoulders and legs	3 cases
Less serious weakness with only slight wasting and little or no restriction of movement	6 cases

Intelligence.

The intelligence of the group as measured by the Wechsler Bellevue Test (adults), and both the W.I.S.C. and Terman (children), is given in Figure I. This shows a distribution which departs somewhat from the normal. It is obvious, however, that if the evidence for mental deterioration were taken into account (see later), this sample would approximate closely to the normal curve.

Mental Deficit.

Mental deficit was found in 50% of the patients. In some this was only slight, and it is possible that with time complete recovery will follow. Table II describes the various degrees of mental deficit under the heading of "mental deterioration", but this does not imply that the process is irreversible.

TABLE I.
Age Distribution of Patients.

Age Group. (Years.)	Number of Patients.
0 to 5	5
6 to 10	7
11 to 15	1
16 to 20	—
21 to 25	—
26 to 30	1
31 to 35	1
36 to 40	1
41 to 45	1
46 to 50	1
51 to 55	—
56 to 60	—
61 to 65	1[1]
66 to 70	2
Total	20

[1] One patient could not be traced.

Memory Defects.

The importance of differentiating between immediate and remote memory loss in cases of organic involvement has been frequently noted (Henderson and Gillespie, 1946). Tests were used in order to detect any difficulty with immediate recall, also any defects or gaps in memory for past events. As the results of Table III show, the defects of immediate memory are greater and are maintained longer.

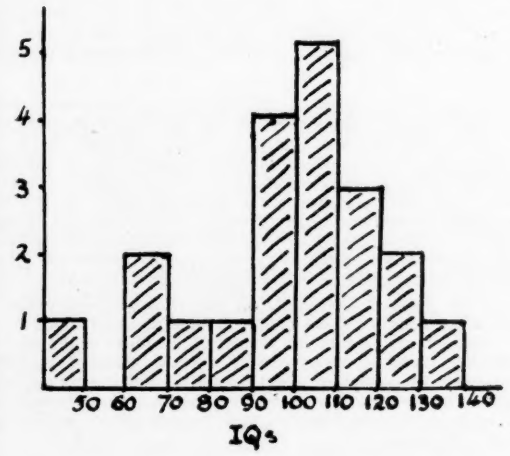

FIGURE I.
Distribution of I.Q.'s.

Amnesic Aphasia.

Inquiry of parents and relatives revealed word loss and semantic difficulty up to three months after the illness and beyond. Table IV sets out the numbers of patients suffering from amnesic aphasia. It indicates that of the 80% of those who had this disability at three months, only 30% were displaying it at 12 months. It will be noted that there were still signs of this difficulty at the time of examination in 30% of the cases.

TABLE II.
Mental Deterioration.

Degree of Deterioration.	Number of Cases.
No deterioration	10
10% to 20% deterioration	7
30% deterioration	2
Over 30% deterioration	1
Total	20

Separation Anxiety.

The questioning of parents revealed that nine of the 13 children showed separation anxiety. This would not have been unusual, except for the fact that the symptoms in some cases were of an extreme kind. For example, one child, aged four years, for several weeks preferred her aunt and would have nothing to do with her mother. Another child, aged six years, for one week disclaimed her siblings and would not let her mother out of her sight. One boy, aged six years, at the sight of the doctor ran away and was only with great difficulty persuaded to cooperate with the examiner.

TABLE III.
Memory Defects.

Memory.	Number of Cases.
Immediate memory:	
Defect maintained up to 3 months	14
Defect maintained up to 6 months	9
Defect maintained up to 12 months	9
Defect maintained up to 18 months	9
Remote memory:	
Defect maintained up to 3 months	7
Defect maintained up to 6 months	4
Defect maintained up to 12 months	1
Defect maintained up to 18 months	1

TABLE IV.
Amnesic Aphasia.

Duration of Symptoms after Illness.	Number of Cases.
Up to 3 months	16
Up to 6 months	10
Up to 12 months	6
Up to 18 months	6
Total	38

In view of the earlier observations that children who have suffered from encephalitis have been refractory and delinquent, together with the fact that there is now good evidence that such behaviour is more common in children separated from their parents, it will be seen that these findings are of some importance for prophylaxis.

Dependence.

Separation anxiety was also associated with extreme dependence, tension, irritability and restlessness. One child, because of his hyperactive behaviour and irritability,

already presented a problem at school. Among both children and adults the severe illness had produced a dependent attitude, which persisted sometimes up to a period of 12 months after the illness. This dependent attitude was shown by eight children and four adults.

Tension and Restlessness.

Tension, issuing in irritability and restless hypermotor activities, remarkably different from the former (pre-encephalitis) personality, was observed in more than half the number of both children and adults. The restlessness took peculiar forms in some children. For example, one child spent time in continuous roaming through the house opening and closing drawers at random. Another child repeatedly dressed and undressed. Most children were fidgety, and some children reverted to nail-biting and thumb-sucking.

In adults the tension gave rise to temper outbursts, nagging and irritability, and some restlessness, which they reported was followed by a feeling of weakness.

Table V indicates the gradual diminution in tension and restlessness.

TABLE V.
Tension and Irritability.[1]

Duration of Tension and Irritability after Illness.	Number of Cases.
Up to 3 months	10
Up to 6 months	11
Up to 12 months	8
Up to 18 months	5
Total	34

[1] Hypermotor activity, considered separately from irritability, was maintained up to six months after illness in 13 cases and up to 12 months after illness in nine.

General Adjustment.

The sequelæ to the encephalitis not unnaturally affected the social adjustment of the patients; but on superficial examination this did not appear to a marked degree. However, a careful examination of the school situation of the children, together with test results, revealed more maladjustment, and the careful probing by psychological tests of the adults was even more revealing.

Table VI gives the results, which show that, while superficially the majority of patients were socially adjusted,

TABLE VI.
General Adjustment.

Adjustment.[1]	Number of Cases.
Social adjustment (general examination):	
Satisfactory	17 } 20
Unsatisfactory	3 }
Educational adjustment and progress (school age patients):	
Satisfactory	6 } 10
Unsatisfactory	4 }

[1] Psychoneurotic symptoms (as shown by Cornell and 16 P.F. tests) present in nine cases.

nevertheless four out of six children were retarded and maladjusted at school, while nine adults showed some psychoneurotic symptoms. Without further follow-up studies it cannot be determined whether these features are temporary or permanent.

Conclusion.

Without attempting any kind of prognosis, the survey revealed that more than half the patients were displaying various degrees of mental deficit, from mild to more severe, and personality disturbances which affected social adjustment in many cases.

Acknowledgements.

The writer desires to thank Dr. E. C. Dax and Dr. C. R. D. Brothers, of the Victorian Mental Hygiene Authority, for their encouragement, and especially Dr. W. J. Stevenson, of the Department of Health, who assisted in locating the cases. The writer is also indebted to the superintendents of hospitals, local health officers and general practitioners, who made records available and assisted generally.

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IT is generally assumed that the serum cholesterol level increases with advancing age and in patients suffering from coronary heart disease (Editorial, 1955; Riegel, 1955). It is also held that in atherosclerosis a reduction of albumin tends to occur even without concomitant hypercholesterolæmia, and furthermore, it is asserted that the protein fractions are intimately involved in the mechanism of lipid clearing (Florey, 1955). Some more specific investigations have shown that changes in the serum globulins follow myocardial infarction (Kuschera and Rettenbacher, 1955).

In the present investigation an attempt was made to determine whether some of the foregoing conclusions could be demonstrated in blood serum of patients diagnosed as suffering from cerebro-vascular disease without manifest symptoms of coronary heart trouble. At the same time, similar tests were carried out on persons of advanced age but in apparent good health, and the results of both groups were compared.

Methods.

Fifty male patients suffering from atherosclerotic cerebro-vascular disease were chosen at random from persons admitted to Lidcombe State Hospital. Cerebral hæmorrhages and thromboses, recent and old, were taken as criteria of vascular lesion in the central nervous system. None of the patients so affected showed symptoms other than those related to possible atherosclerotic changes. Thus the other causes of cerebral hæmorrhage, such as syphilis, *periarteritis nodosa* and blood dyscrasias, were excluded. The following sequence of examinations was observed. Serum cholesterol levels were determined by the Liebermann-Burchard reaction (Kenny, 1952); blood pressure values, age, number of accidents and the course of the

disease were noted. This was done with a view to correlating the serum cholesterol levels with the outcome and prognosis of the cerebro-vascular accident. Further, certain tests for the determination of serum protein patterns were carried out. In the absence of the elaborate equipment needed for ultracentrifuge observations and electrophoresis, some simple liver function tests were selected which can be carried out as a routine measure in minor hospitals. The thymol turbidity, zinc turbidity, thymol flocculation and cephalin cholesterol flocculation tests were considered as most appropriate. It is thought that the mechanism of all these tests consists of quantitative and qualitative changes in the albumin and globulin fraction of the serum proteins responsible for the flocculation—*i.e.*, turbidity—evidenced (Miller, 1952). There is also evidence that some of the lipids associated with the beta globulins of serum increase turbidity formation.

The same battery of tests was performed on 50 men of advanced age, whose general condition did not reveal any changes beyond those consistent with the physiological process of aging.

Results.

The summarized results are set out in Tables I, II and III.

TABLE I.

Distribution of Serum Cholesterol Levels in 50 Patients with Cerebro-Vascular Disease and in 50 Healthy Old Men.

Subjects.	Serum Cholesterol Levels. (Milligrammes per 100 Millilitres.)			
	100–149	150–199	200–249	250–300
50 patients with cerebro-vascular disease	1	8	33	8
50 healthy old men	7	33	9	1

Although it was found that in 82% of the patients suffering from a cerebro-vascular accident the serum cholesterol level exceeded 200 milligrammes per 100 millilitres, nevertheless no relationship was established between the differences in the increased serum cholesterol levels and the course of the accident. From Table I it is evident that the serum cholesterol level of healthy old people is less than 200 milligrammes per 100 millilitres in 80% of the present cases.

TABLE II.

Comparison of Blood Pressure, Age Groups and Serum Cholesterol Levels in 50 Healthy Old Men and 50 Patients with Cerebro-Vascular Disease.

Age. (Years.)	Serum Cholesterol Level. (Milligrammes per 100 Millilitres.)			
	100 to 199.		200 and Higher.	
	Normal Blood Pressure.	Hypertension.	Normal Blood Pressure.	Hypertension.
Healthy Old Men (50 Subjects).				
75 to 84	17	4	4	—
85 to 94	15	2	5	—
95 and older	2	—	1	—
Patients with Cerebro-Vascular Disease (50 Subjects).				
50 to 64	—	4	4	3
Over 65	3	3	21	12

The joint results of blood pressure readings and serum cholesterol estimations are given in Table II. In the present series no healthy subject had both serum cholesterol levels exceeding 200 milligrammes per 100 millilitres and hypertension. It is to be noted that in our series the number of hypertensives is not increased among those affected by a cerebro-vascular accident. In this investigation a diastolic blood pressure reading of over 100 millimetres of mercury was considered as indicative of hypertension (Table II). As to the altered serum protein patterns, no evidence supporting this observation can be derived from the thymol turbidity, zinc turbidity and thymol flocculation tests. However, the large number (79 out of 100) positive results to cephalin cholesterol tests in both healthy and diseased subjects is remarkable; more than 50% of the positive results were "++" (Table III).

Comment.

There is no consensus of opinion regarding the upper limit of normal serum cholesterol values. In the United States of America a figure up to 310 milligrammes is considered as normal, although most workers in this field give 260 milligrammes per 100 millilitres as the normal maximum (Editorial, 1956). Moreover, the American normal levels cannot be used as universal standards, as they are excessively high, possibly owing to the fat-rich diet of the American population (Page, 1957). On the other hand—*e.g.*, in the Japanese and South Africans—the levels are much lower and coronary disease is less prevalent. Therefore, the difficulties of demonstrating an association of serum lipids with atherogenesis in the United States of America are possibly due to the widespread incidence of this disease in that population (Gofman, 1956). Whether this deduction is also applicable to the Australian community remains to be investigated. To my knowledge, no representative figures for cholesterol values are available for Australia.

TABLE III.

Results of Thymol Turbidity, Thymol Flocculation, Zinc Turbidity and Cephalin Cholesterol Flocculation Tests in Cerebro-Vascular Disease (50 Patients) and Healthy Old Men (50 Subjects).

Subject.	Thymol Turbidity, Thymol Flocculation and Zinc Turbidity Test.		Cephalin Cholesterol Flocculation Test.	
	Result Abnormal.	Result Normal.	Result Abnormal.	Result Normal.
Patients with cerebro-vascular disease	3	47	40	10
Healthy old men	—	50	39	11

One significant conclusion has been brought home by the recent nation-wide cooperative survey on lipoproteins in the United States—namely, the fact that there is no advantage in using the more elaborate and time-consuming methods (ultracentrifuge testing, *etc.*) as against the cheaper and simpler serum cholesterol determination, which is equally conclusive. Moreover, it is generally assumed that lipid metabolic abnormalities are not the only cause of atherosclerosis, but other factors are also contributory.

Although both the thymol turbidity and cephalin cholesterol tests measure alteration in the serum protein pattern, in our series the results of these tests showed a marked discrepancy. One common denominator of both groups examined was advanced age, which can possibly be offered as an explanation for the 79% of positive results to the cephalin cholesterol test.

Also it is to be noted that 82% of the healthy subjects had serum cholesterol levels below 200 milligrammes per 100 millilitres. The significance of this circumstance will be followed up in a further study on a larger number of subjects.[1]

Summary.

1. The serum cholesterol level of 50 male patients with cerebro-vascular disease was compared with that of 50 healthy men of advanced age.

[1] This observation has since been corroborated by the figures of a subsequent, still current investigation.

2. It was found that in 82% of the healthy old men the serum cholesterol level did not exceed 200 milligrammes per 100 millilitres.

3. A battery of liver function tests was performed to determine possible changes in the serum protein pattern of both groups. Whereas 79% of all tested subjects gave a positive response to the cephalin cholesterol test, the number of other liver function tests which produced abnormal results was negligible.

Acknowledgements.

I am grateful to Dr. H. G. Wallace, the Director-General of Health, New South Wales, and to Dr. G. S. Procopis, Medical Superintendent, Lidcombe State Hospital, for permission to publish the results of this investigation. To Dr. Procopis my thanks are also due for checking the manuscript. I am also indebted to the staff of the microbiology laboratories of the New South Wales Department of Public Health and the pathology department of Lidcombe State Hospital for their continued cooperation.

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OBSERVATIONS ON SERUM ANTIBODIES IN ABORIGINES OF THE NORTHERN TERRITORY.

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AND

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IN August, 1956, the Wenner-Gren Anthropological Foundation supported an expedition to the Northern Territory. During the expedition blood was taken from aborigines of six tribes in the neighbourhood of Haast's Bluff.

Owing to a continuation of previous interest in Murray Valley encephalitis (Miles and Howes, 1953) and "Q" fever (Beech, Howes and Miles, 1953; Dane and Beech, 1954-55) among aborigines in the Northern Territory, the sera collected on this occasion were tested for antibodies to the agents of the two diseases. Also, as it is known that yaws occurs among the aborigines, serological tests for syphilis were carried out, as these are known to give positive results with sera from people with yaws. In addition, during the examination of the sera it was noted that eosinophilia occurred frequently (Casley-Smith, 1957), so the small number of sera still available were tested for their ability to fix complement in the presence of a hydatid antigen. This paper describes the results obtained.

Materials and Method.

Sera.

The sera used in this investigation were obtained from 50 aborigines, comprising 39 males and 11 females. The ages ranged from 10 to 75 years. Most (32) of the aborigines were aged between 21 and 45 years. Twenty-seven were younger than 36 years and the remainder (23) were older.

Murray Valley Encephalitis.

Complement fixation tests were carried out by the method described by Miles and Howes (1952).

Neutralization tests were carried out by the screening method described by Warner (1957), and those sera neutralizing 20 LD_{50} of virus were designated "positive".

"Q" Fever.

Complement fixation tests for "Q" fever were carried out according to the method described by Beech, Howes and Miles (1953), except that overnight fixation at 4°C. was used in place of one hour at 37°C.

Serological Tests for Yaws (Syphilis).

Serological tests for yaws and syphilis were carried out by the Bacteriology Division of the Institute of Medical and Veterinary Science. All sera were first tested by the Kline test, and those giving positive results were further tested by the Wassermann and Kahn tests.

Hydatid Complement Fixation Tests.

These were also carried out by the Bacteriology Division of the Institute, using an antigen consisting of fluid from a hydatid cyst.

Results.

Murray Valley Encephalitis.

Of 50 sera tested by the complement fixation test, 11 were anticomplementary. Of the remaining 39, none fixed complement of a dilution of 1:5. Three out of 27 sera tested by the neutralization test were "positive".

"Q" Fever.

Of the 36 suitable sera, one fixed complement at a dilution of 1:20. The donor of the serum was a male, aged 32 years, from the Ngalia tribe. The remainder of the sera failed to fix complement at a dilution of 1:5.

Yaws.

Nine out of 50 sera gave positive results to the Kline test, and of them only four gave positive results in the Kahn and Wassermann tests. Three of the "positive" sera were from males and one from a female. All were aged more than 38 years.

Hydatid Disease.

Of 10 sera tested, two were anticomplementary, while none of the remaining eight fixed complement in the presence of hydatid fluid antigen. Two of the sera on which satisfactory tests were made came from natives with 4% and 5% eosinophilia.

Discussion.

The incidence (three out of 27 tested) of Murray Valley encephalitis (MVE) neutralizing antibodies found in the sera described here shows no evidence of being different from that (11 out of 40 tested) reported by Miles and Howes (1953) in sera obtained at least three years previously from the same area. The only conclusion that can be drawn from this is that there has been no considerable dissemination of MVE virus in the area during the period between the surveys.

The interest in "Q" fever lies in the demonstration of antibodies in three out of five kangaroos in the Northern Territory and the north of South Australia (Dane and Beech, 1954-55), following on the discovery that six of 328 (1·8%) of sera from aborigines in the Northern Territory fixed complement with "Q" fever antigen (Beech, Howes and Miles, 1953). Our results (one positive result out of 36 sera tested) are of the same order. However, these results do not differ very markedly from those obtained with sera from white people in South Australia (one positive result out of 304 sera tested). Hence, even if it is found that a large proportion of kangaroos are infected with *Coxiella burneti*, they do not appear to spread the organism to any extent among the human population in the Northern Territory.

Four positive Wassermann reactions out of a total of 50 sera tested suggests that in the entire population the incidence of "positive" sera lies somewhere between 2% and 19%. Since syphilis is probably rare among aborigines in this part of the Northern Territory, these results indicate the possible extent of infection of the aborigines with yaws.

An incidence of four positive Wassermann results out of 50 sera tested is lower than that (nine out of 33) obtained by Miles (unpublished results) in sera collected in 1951 from the same area. Thus the incidence of "positive" sera may be diminishing, which may be a reflection of the increased use of penicillin in recent years. In this connexion, although it may be a matter of chance, all four "positive" sera tested by us were obtained from aborigines aged more than 38 years. This may have resulted from the tendency for younger natives to make greater use of medical treatment than the older ones.

It was thought that hydatid disease could be widespread among the natives, as they lived in close contact with innumerable dogs which often fed on the raw remains of slaughtered cattle. This suspicion was heightened by the discovery of eosinophilia among the natives (Casley-Smith, 1957). However, none of the eight sera tested fixed complement with hydatid fluid, and two of them came from natives with eosinophilia. This suggests that, if hydatid disease does occur, less than one-third of the natives are infected, and that the eosinophilia should probably be attributed to some other cause.

Summary.

Fifty aboriginal sera were obtained from the Yuendumu area in the Northern Territory. Some or all of the sera were tested for evidence of infection with Murray Valley encephalitis (MVE), "Q" fever, yaws and hydatid disease.

There was no evidence that the incidence of MVE neutralizing antibodies (three of 27 sera) had changed since a survey of the area several years previously. The incidence of "Q" fever antibodies (one of 36 sera) was of the same order as that obtained previously among aborigines in the Northern Territory, and as that of white people in South Australia. Four of the 50 sera yielding positive Wassermann reactions suggested that approximately anything up to one-fifth of the total population had been infected with yaws at one time or another. None of eight sera contained antibodies against hydatid disease.

Acknowledgements.

We gratefully acknowledge the painstaking technical assistance of Brian Moore. We are indebted to the Bacteriology Division of the Institute of Medical and Veterinary Science for carrying out the tests for yaws and hydatid disease.

The material on which this paper is based was collected on a University of Adelaide anthropological expedition financed mainly by grants from the Wenner-Gren Foundation for Anthropological Research, New York, and the University of Adelaide. We are indebted to Professor A. A. Abbie, who led the expedition, for making this material available. We are also indebted to Professor J. B. Cleland for suggestions and helpful discussion.

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Reports of Cases.

IMMUNIZATION OF Rh-NEGATIVE POST-MENOPAUSAL WOMEN BY Rh-POSITIVE BLOOD TRANSFUSIONS.[1]

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As a result of the shortage of Rh-negative donors, Rh-positive blood has frequently to be used for Rh-negative male patients and for female patients beyond the child-bearing age. This procedure holds for patients of both sexes who have not been immunized by previous Rh-positive blood transfusions, and also for women of this age group who have not been sensitized by pregnancies in which the child was Rh-positive.

Since this change has become necessary, three women are known to have been sensitized in the Royal Women's Hospital, Melbourne.

Case I.

A., aged 48 years, was first tested in July, 1955. She was found to have group A, Rh-negative blood containing no Rh antibodies in albumin tests. Blood cells from four Rh-positive donors were cross-matched against her serum by the saline and albumin techniques at room temperature and 37·5° C., and by the indirect Coombs test. No incompatibility was detected. This blood was given without obvious reaction. She was readmitted to hospital on March 29, 1956, with a hæmoglobin concentration of only 50%. Red blood cells from four Rh-positive donors were cross-matched against her serum by the saline, albumin and indirect Coombs techniques. All were found to be incompatible. The albumin titre was found to be 256.

Case II.

B., aged 60 years, was found to be group O, Rh-negative on August 24, 1955, when her serum was cross-matched with four pints of group O Rh-positive blood. All this blood was given without evidence of any reaction. No antibodies were detected in tests carried out on April 24, 1956. At that time she needed a further blood transfusion, and cross-matching tests between the patient's serum and red cells from three donors showed no incompatibility by the albumin and saline techniques at 37·5° C. and room temperature, or by the indirect Coombs test. Blood from the three donors was given without obvious reaction. Twenty-five days later tests revealed the presence of Rh antibodies with a titre of four by the albumin technique.

Case III.

C., aged 40 years, was found to be group O and Rh-negative. No Rh antibodies were detected by the albumin technique. She was given two pints of group O, Rh-negative blood on January 29 and again on January 31, 1955. On February 3 she underwent an operation and was given four pints of group O, Rh-negative blood, followed by six pints of group O, Rh-positive blood. The red blood cells from each of the Rh-positive donors were tested against the serum of the patient by the saline and albumin techniques at room temperature and at 37·5° C. and by the indirect Coombs test. All appeared compatible. The next day the patient was flushed and her temperature was 102° F. Her blood was investigated on February 8, when the temperature was 102° F. and the patient was jaundiced; it was found to contain Rh antibodies with an albumin titre of 64 and a saline titre of 16. The serum bilirubin content was 14 milligrammes per 100 millilitres. She had had no previous blood transfusions, but investigation of her obstetric history revealed that she had a child

[1] This work was made possible by a grant from the National Health and Medical Research Council.

now aged 14 years, and that she had had one miscarriage. It would appear that the patient had suffered an anamnestic blood transfusion reaction, which might have commenced unnoticed during anæsthesia.

Comment.

Three important features are revealed in these cases:

1. The patients were in the middle age group, two being aged under 50 years.

2. There is little knowledge at present concerning the length of time needed for the formation of Rh antibodies. In the second case described, the antibodies were present 25 days after the second series of blood transfusions. They may have been present much earlier. It is possible that they were present in amounts just below the level which can be detected by laboratory tests before the last series of transfusions was given.

3. The danger of anamnestic reactions in women who have had pregnancies years before the present operation is illustrated in Case III.

Discussion.

The possible effects of using Rh-positive blood for patients at this hospital were appreciated at the outset, because of the frequent use of pre-operative transfusions combined with further administration of large amounts of blood during major surgical procedures. The danger to these patients lies in the possibility of reactions which may not be recognized whilst further transfusions are being given to the patient under anæsthesia.

Although the proportion of individuals affected is relatively small, the attendant risks of giving further Rh-positive blood require very great consideration. Cross-matching in such cases must include testing of the patient's serum against donor cells, both in saline and in albumin suspensions, and also by the indirect Coombs technique. Even when such tests indicate that the donor blood is compatible, anamnestic reactions such as the one observed in Case III can occur if antibodies are present in amounts below the level which can be detected by laboratory tests.

A CASE OF INTESTINAL OBSTRUCTION FOLLOWING THE ADMINISTRATION OF PENTAPYRROLIDINIUM BITARTRATE ("ANSOLYSEN").

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THERE is today quite a wide use of ganglion-blocking compounds in the treatment of hypertension, and of these, pentapyrrolidinium bitartrate ("Ansolysen") is the commonest. Its side effects, such as dizziness and constipation, are well known. In the present case is reported the rapid onset of ileus simulating acute intestinal obstruction while the patient was undergoing treatment for hypertension with "Ansolysen".

Clinical Record.

A male patient, aged fifty-eight years, was admitted to hospital at 4 p.m. on August 6, 1956, complaining of abdominal pain and vomiting present for forty-eight hours.

He said that he had been well until 1944 (then aged forty-six years), when he was discharged from the Army with hypertension. Since then he had been in hospital three times, once with severe dermatitis and twice with shortness of breath. Since his last admission to hospital two years previously he had been taking reserpine ("Serpasil"), 0·25 milligramme twice daily. He also had had slight indigestion some years previously, but none in the last four years. One year prior to his admission to hospital he had an attack of abdominal pain requiring the injection of an analgesic for its relief. He did not remember what the pain was like, but thought it might have been similar to the present attack, but it lasted for only a few hours. Over the last ten years he had had increasing shortness of breath, and for the last two years this had almost incapacitated him. Three weeks prior to his admission to hospital his local doctor commenced treating him with "Ansolysen", starting with one 40 milligramme tablet per day and increasing the dosage to 40 milligrammes four times a day, the latter dosage being reached about four days prior to his admission to hospital. The patient said that his local doctor had told him that his blood pressure varied between 200 and 240 millimetres of mercury, systolic, before he began on the "Ansolysen" therapy.

On the afternoon of August 4 he had taken rather more exercise than was customary, but felt well apart from his usual shortness of breath. After a normal evening meal he was sitting by the fire when he developed severe lower abdominal pain, which was colicky in nature, doubled him up and made him vomit. This pain he described as all over the lower part of his abdomen, below the umbilicus, and it did not radiate. In between the attacks of pain, which came approximately every five minutes, he felt nauseated and uncomfortable, but not in pain. He called his local doctor, who gave him an analgesic by injection and told him that his blood pressure was only 80 millimetres of mercury, systolic, and that he would therefore have to stay in bed. An enema was administered which produced a good result. After that he had no more pain for twenty-four hours, but he continued to vomit occasionally and he noticed gradual swelling of his abdomen. On the afternoon of August 5 he had a further attack of colicky pain, and he was given a further injection of analgesic which rapidly relieved it. After that, and prior to his admission to hospital on August 6, he had no further pain; but he occasionally vomited brown fluid and he noticed increasing abdominal distension. He had last vomited four hours before his admission to hospital. Up to the onset of his abdominal pain he had had a regular bowel action once a day, including the morning of August 4; but after the onset of the pain, apart from the result of the enema, he had passed neither flatus nor fæces. During the twenty-four hours prior to his admission to hospital he had passed only a small amount of urine on only one occasion, although he had continued to drink fluids in between bouts of vomiting. He said that he felt well, and that his breathlessness since the onset of the pain had been less than it had been for years.

Examination of the patient revealed him to be a flushed, elderly man, lying quietly in bed in no distress. His skin felt warm and dry, and there was a slight loss of subcutaneous elasticity; his fingers were warm and his nails were pink. His pulse was regular, but of poor volume, at a rate of 84 per minute. His blood pressure was 90 millimetres of mercury, systolic, and 70 millimetres, diastolic. His jugular venous pressure was not raised. His tongue was dry and coated and his breath offensive. His chest moved poorly with respiration and was comparatively fixed in the inspiratory position. His chest was resonant to percussion in all areas; breath sounds were very faint, and no adventitiæ could be heard. His apex beat was impalpable, and no cardiac dullness was elicited on percussion. His heart sounds were regular and faint, and no bruits could be heard. His abdomen was distended, but not tense. There was no muscle guarding or rigidity. Slight generalized tenderness was elicited on deep palpation, but no release tenderness. No mass or viscus was palpable. On percussion, his abdomen was resonant anteriorly and dull in the flanks, but there was no shifting dullness. Borborygmi were audible on auscultation. Rectal examination revealed that his rectum was empty. His prostate was slightly enlarged, but otherwise normal, and there was no tenderness. There was a little brown fluid on the glove. A plain X-ray film of his abdomen revealed some distended loops of small bowel with some fluid levels. An electrocardiogram revealed left heart strain. The serum electrolyte contents were normal. A diagnosis of bowel obstruction of unknown ætiology was made.

Intravenous therapy, consisting of the administration of a litre of normal saline followed by a litre of 4% dextrose

solution in one-fifth normal saline, was ordered, and a tube was passed into the stomach, from which eight ounces of brown fluid were aspirated. Four hours after his admission to hospital his pulse rate was 80 per minute and his blood pressure 100 millimetres of mercury, systolic; he commenced to pass some fluid fæces and a large amount of flatus *per rectum*. At this stage no more fluid had been aspirated from the stomach. Eight hours after his admission to hospital (at midnight) his pulse rate was 84 per minute and his systolic blood pressure 120 millimetres of mercury. During the early hours of the morning he had five fluid bowel actions, and only a further two ounces were aspirated from his stomach.

By 10 a.m. on August 7 he said that he felt well and hungry. His pulse rate was 76 per minute, and his blood pressure was 160 millimetres of mercury, systolic, and 100, diastolic. The abdominal distension was relieved. Intravenous therapy was discontinued after the total of two litres originally ordered had been given, and he commenced to take fluids by mouth. By 4 p.m. his blood pressure was 200 millimetres of mercury, systolic, and 110, diastolic, and his pulse rate was 76 per minute. His abdomen was lax and not tender on deep palpation. A further plain X-ray film of his abdomen revealed considerably less distension of the small bowel, with most of the air now in the colon and a minimum of fluid levels.

He was observed for one week, and no further attacks of pain occurred. His bowels opened regularly once a day, and his blood pressure remained stable at 210 millimetres of mercury, systolic, and 115, diastolic.

Discussion.

This man was treated conservatively because, although he had clinical obstruction for forty-eight hours with a systolic blood pressure below 100 millimetres of mercury, he was not suffering from shock in the clinical sense, in that his extremities were warm and red. Subsequent interrogation revealed the history of taking "Ansolysen", although at the time of his admission to hospital this was not known.

Constipation and occasionally ileus are well recognized complications of the administration of "Ansolysen" and other ganglion-blocking agents; but search of the literature on the subject has failed to reveal any record of ileus simulating acute intestinal obstruction. Gifford, Allen and Birkhead, at the Mayo Clinic, in 1953 reported, in a series of 12 patients treated with "Ansolysen", one man, aged sixty-one years, who developed paralytic ileus after only one week's treatment. This subsided with only conservative management. Of the remaining 11, 10 needed a laxative to relieve some degree of constipation.

Grimson and Durham (1955), in a review of drugs used in the treatment of hypertension, report in a series of 14 patients troublesome fluctuations in blood pressure due to delayed gastric emptying. The need for spacing the doses of "Ansolysen" is now well known. Of 37 patients taking hexamethonium, 21 developed some degree of constipation and seven had epigastric discomfort. Other writers all report varying degrees of constipation and occasionally ileus. Goldsmith *et alii* (1954) record one death from ileus in a patient undergoing treatment for hypertension with hexamethonium bromide.

In the present case, it is presumed that there was a build up of "Ansolysen" in the upper part of the alimentary tract, due to delayed gastric emptying, and this accounted for the sudden collapse from hypotension. At this stage ileus developed, complete in the lower part of the bowel, but only partial proximally; this accounts for the attacks of colic after ingestion of food and then fluid.

Acknowledgements.

My thanks are due to the chairman of the Repatriation Commission for permission to publish this case, and to Dr. H. Phillips, visiting surgeon to the Repatriation General Hospital, Heidelberg, who was in charge of the patient.

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A CASE OF INFANTILE GLAUCOMA ASSOCIATED WITH MATERNAL RUBELLA.

By KEVIN O'DAY,

Melbourne.

THE following case is of interest for two reasons: (i) The mother was seen by her family doctor "with a pretty typical clinical rubella three weeks after missing a period *id est* about 6-7 weeks of pregnancy". (ii) The association of rubella with infantile glaucoma is uncommon.

Clinical Record.

Baby A., a boy, aged six weeks, was referred to me on August 16, 1956, by Dr. G. Forristal, of Sunshine, with bilateral infantile glaucoma. There were four other boys in the family, and Dr. Forristal had been treating them for rubella when the mother contracted the disease herself. This occurred three weeks after she had missed a menstrual period, as she mentioned to Dr. Forristal at the time. The rest of the pregnancy was uneventful and labour was normal.

On examination of the baby, the corneas were found to be enlarged, the corneal epithelium was œdematous, diffuse opacities were already present in the corneal parenchyma, and the pupils were semi-dilated. No clear view was obtained of the fundi. Eserine-salicylate drops (0·25%) were ordered for four-hourly instillation into each eye. On August 20, the pupils were small, but there was no evidence of decrease in tension in either eye. On August 21, the little patient was examined under general anæsthesia at St. Vincent's Hospital, Melbourne. The intraocular tension was at least 30 millimetres (Schiötz) in each eye, and the corneal diameter was approximately 14 millimetres in each eye. After a preliminary external canthotomy on the left eye, a goniotomy was performed on the left eye by the method of Barkan.

On August 28, under general anæsthesia, the tension in the right eye was 36 millimetres (Schiötz) and in the left eye 15 millimetres (Schiötz). The appearance of the right cornea had not changed; but the œdema of the left corneal epithelium had cleared, although the deeper opacities were still present. After an external canthotomy, a goniotomy was performed on the right eye. As the knife-needle swept round the filtration angle, it became entangled in the iris, which was torn and bled a little.

Both operations were performed without the aid of a contact glass. Although the point of the knife is hidden by the opaque tissue at the limbus, it can be guided into the filtration-angle without any great difficulty, and the gritty sensation which is felt as it engages the tissues of the angle in its sweep downwards is sufficient assurance to the surgeon that he is the correct plane. Barkan recommends that the blade should be tilted forwards a little to prevent engagement with the iris. Even if this does happen, apparently no great harm is done. In this situation the edge of the lens is well behind the iris and is not likely to be

damaged. The resulting hyphæma is quickly absorbed. This little accident has happened in my practice on two occasions, with no harm resulting.

Convalescence was uneventful, and tension (estimated with the Schiötz tonometer under general anæsthesia) has remained normal in each eye. The deeper opacities in the cornea have disappeared, and the normal fundi are now easily visible.

Associated with the ocular defect is a patent *ductus arteriosus* which so far has not prevented the baby from thriving.

The mother's attack of rubella occurred about the sixth to the seventh week of pregnancy. Whilst at this time the lens is in the very early stages of differentiation, the tissus which will enter into the formation of the filtration angle give no indication of their presence. If the virus of rubella was responsible for the aberration causing the development of glaucoma, one would expect a much greater deformity in all the tissues of this region, and also involvement of the iris, the ciliary body and the lens.

Discussion.

Whilst it seems too much hope for permanent relief from such an apparently intractable condition by a comparatively simple operation, there is no obvious contraindication to its use as early as possible in infantile glaucoma. In the following case the tension has remained normal in each eye for nearly four years. The deeper corneal opacities have almost completely cleared, no photophobia is present, and whilst as yet no accurate estimation of the visual acuity is possible, the child plays quite normally with her nine brothers and sisters.

Baby B., aged six months, was referred to me on September 4, 1953, by Dr. T. Greening of Ballarat. On September 7, and September 14, goniotomy was performed on the right and left eye respectively. On November 25, 1954, the operation was repeated on the left eye, as the tension, which was checked every three months under general anæsthesia, had risen a little. There has been no further rise of tension in either eye.

Summary.

1. A description has been given of a case of infantile glaucoma which was associated with an attack of maternal rubella in the early months of pregnancy.

2. Attention has been drawn to the value of the operation of goniotomy in infantile glaucoma, and to the comparative ease with which it may be performed.

Reference.

BARKAN, O. (1948), "Technic of Goniotomy for Congenital Glaucoma", *Tr. Am. Acad. Ophth.*, 52: 210.

Reviews.

Handbook on Poliomyelitis. By Joseph Trueta, M.D., D.Sc. (Hon.), (Oxon.), F.R.C.S. (Hon.) (Canada), F.R.C.S., Nuffield Professor of Orthopædic Surgery, University of Oxford, A. B. Kinnier Wilson, M.A., M.B., M.R.C.P., D.P.M., and Margaret Agerholm, M.A., B.M., B.Ch. (Oxon.); 1956. Oxford: Blackwell Scientific Publications. 9" × 5¾", pp. 147, with illustrations. Price: 20s.

DESPITE the infrequency of acute poliomyelitis in the last few years, this book makes interesting reading. It is short and very much to the point, and the authors obviously have had much experience of the disease. The introductory chapters cover ætiology, pathogenesis, pathology, clinical features, diagnosis and prevention and are a useful summary of modern views. Treatment during the acute stage is covered, the authors advocating the carrying out of passive movements short of pain aided by packs frequently from the beginning. There is a well-balanced discussion of the Kenny treatment. Such important points as maintenance of normal posture, prevention of pain, œdema and pressure sores, urinary retention, constipation and diet are dealt with thoroughly. Respiratory complications, their causes and clinical features are well summarized. The chapter on their management contains little physiological detail and few complete descriptions of mechanical apparatus, but has many helpful points on management and nursing. As far as muscle weakness is concerned, the authors do not recommend early vigorous active movements. They think that a period of one to eight weeks follows the acute stage in which there may be persisting meningism, lethargy, irritability, tiredness and abnormality of the cerebro-spinal fluid. During this stage local and general fatigue is absolutely prohibited. This avoidance of fatigue is again emphasized in the stage of recovery of muscle power, a view which is the subject of controversy. The difficulties of "weaning" the patient from the respirator are dealt with well. The text concludes with a short discussion of the management of permanent disability by apparatus and surgery and some very pertinent remarks on rehabilitation. The book is well produced, with good line drawings and photographs. There is an adequate bibliography; the index is excellent. It can be recommended to anyone concerned with the management of this disease.

Unexpected Reactions to Modern Therapeutics: Antibiotics. By Leo Schindel, M.D.; 1957. London: William Heinemann (Medical Books), Limited. 7¼" × 5", pp. 160. Price: 15s.

IT is estimated that nearly one and a half million lives were saved in the United States alone during the period 1938-1952 by the use of antibiotics and sulphonamides. Considering the enormous consumption of these drugs today, the toxic side effects are very few indeed; but for this same reason few clinicians will not have encountered such reactions. This book is a compilation of the "undesirable, unexpected and annoying" responses to drugs, which at times may cause the physician some anxiety in wondering whether the end justifies the means. The author has presented information, which is usually only to be found by a time-consuming literature survey, in a concise form which will be of value and interest wherever these drugs are used.

The book is divided into 12 chapters dealing with penicillin, streptomycin, chloramphenicol, the tetracyclines, antibiotic resistance, neomycin, erythromycin, bacitracin, fumagillin, novobiocin, cycloserine and polymyxin. Each drug is briefly discussed in an historical opening to the chapter, and this is followed by a description of the therapeutic uses and common side-effects. Then follows a detailed description of special reactions which have been described for that particular drug, together with a list of the principal references which are of value.

The author is to be congratulated on such a concise and informative book, which should have a wide appeal.

Biochemical Disorders in Human Disease. Edited by R. H. S. Thompson, M.A., D.M., and E. J. King, Ph.D., D.Sc., F.R.I.C.; 1957. London: J. and A. Churchill, Limited. 9¾" × 6¼", pp. 858, with 121 illustrations. Price: 90s.

THIS trans-Atlantic collaboration, envisaged some years ago, has at last been published, simultaneously in England and America. There are 20 chapters representing the effort of 31 contributors, of whom 21 are British, the remainder American, under the general editorship of two British contributors. The book had its impetus from the increasing, unfortunate interdependence of the fields of biochemistry and medicine. Its purpose is to set before medical readers the way in which biochemistry now impinges on medicine and surgery; in this way problems are also put before biochemists—this, it is hoped, will help them understand the needs of present-day clinicians. The approach differs somewhat from chapter to chapter, depending both upon the material and upon the author's individuality. In this way the book retains the freshness of a symposium.

The chapters are selected on an "organ basis", which is particularly suitable for the book's purpose. They deal with some aspects of disease within most of the major systems, some naturally lending themselves to discussion. There is but slight overlap between chapters—*e.g.*, phæochromocytoma is considered in the chapter on hypertension and that on adrenal diseases; aspects of cholesterol metabolism are of necessity discussed in the chapters on *diabetes mellitus* and hypoglycæmia, disorders of nutrition, and lipidoses; some aspects of electrolyte disorders are considered under diseases of the gastro-intestinal tract and also under diseases of the kidney *etc.* Needless to say, the emphasis is upon metabolic aspects of disease—in the widest sense—as reference to the American-style table of contents and chapter headings will confirm.

There is, in general, no detailed description of chemical methods used; reference to methods is mostly in the interests of clarity and to ensure that clinical emphasis is correct. For example, Jailer and Longson discuss steroid methods in their chapter on adrenal diseases, Bywaters and Glynn refer to the specificity of serological changes in rheumatoid arthritis, and Sommerville comments upon methods of hormone assay *etc.* There is no doubt that these

comments should help many in assessing the usefulness and limitation of perhaps too readily accepted techniques.

The selection of authors vouches for the interest in this book; to mention only some, R. G. MacFarlane discusses "The Clotting Mechanism"; M. D. Milne, "Diseases of the Kidney and Genito-Urinary Tract"; C. H. Gray, "Porphyrias"; Dorothy Andersen, "Glycogen Storage Diseases and Galactosæmia"; J. W. Jailer and D. Longson, "Adrenal Diseases"; S. J. Thannhauser, "Lipidoses", *etc.* Each chapter ends with a selected list of references, mostly up to 1955. There is a full author and subject index.

This book is in no wise a text-book of biochemistry; the authors aim to show "currents", and while here and there another specialist may decry emphasis and interpretation, within its scope it is of admirable value. It should be in the hands of all thinking clinicians and senior students. It is to be hoped that there will be subsequent editions.

Changes in Liver Cell Elements during Stimulated Protein Synthesis: A Cytochemical Study. By G. F. Bahr; 1957. Stockholm: Acta Radiologica Supplement 147. 9¼" x 7¼", pp. 112, with 59 illustrations. Price: Sw. Kr. 25.

DURING the past few years intensive research has been done on the smaller elements contained in cell nuclei and cell cytoplasm, especially in relation to distribution of cell enzymes and to protein synthesis. It has been recognized for some time that the cell nucleus plays a major part in protein synthesis.

In the present book, G. F. Bahr, working at the Institute for Cell Research, Medical Nobel Institute, Stockholm, has detailed the work done in investigating the changes in protein synthesis in the nuclei and microsomes of liver cells in rats in starving and refeeding experiments, hepatectomy, and precancerous livers treated with paramethylamino-azobenzene.

The determinations were made by physical methods involving the use of a new kind of scanning interference microphotometer, a specific dyeing method for sulphydryl groups with improved microspectrophotometric techniques, and a new method involving the use of the electron microscope to weigh ribonucleoprotein particles. A large part of the book, which contains only 111 pages, is taken up with descriptions of the instruments and of their method of use. The results obtained are given in some detail and are interesting; but the whole book is very highly specialized, and apart from the summary of results, can be of interest only to those engaged in work of a similar character. The book is interesting in showing how the most refined methods of physical measurements can be used in investigating biochemical changes in cells.

The Technique of Fluid Balance: Principles and Management of Water and Electrolyte Therapy. By Geoffrey Tovey, M.D.; 1957. Edinburgh and London: Oliver and Boyd. 8¾" x 5½", pp. 108, with tables.

WHILE at first wondering if there can be a place for so many monographs and text-books on fluid and electrolyte balance, one is at once reassured by a consideration of the tremendous importance of the subject and its complexity. Each author, seeing the problem in his own way, must cater for the needs of a different section of readers.

The present volume is directed to the relatively uninitiated, and is mainly concerned with diagnosis and management at a clinical level. It is written simply and clearly with no embellishments, and diagrams and tables are numerous, orthodox and helpful. After a consideration of the sources of imbalance and of the pathogenesis of resulting symptoms, the discussion passes to the principles and practice of maintaining fluid balance, and then to diagnosis and methods of correction of imbalances. In all sections the approach is essentially quantitative, and calculation of the actual deficits are attempted on the basis of clinical manifestations as well as biochemical data. Techniques and rates of administration and safety measures are precisely indicated.

One wonders whether the author's thinking is quite clear on the mechanism of acid-base disturbances, for it is certainly misleading to attribute such disturbances to changes in sodium or chloride ion; it would be better to indicate that these merely reflect movement of hydrogen ion, and that it is the latter alone which can effect hydrogen ion concentration. Thus it is not the loss of chloride but of hydrochloric acid which leads to the alkalosis of vomiting. In view of the present intensive search for the location and effector mechanism of the volume receptor, it is surprising to read repeatedly that, when volume control takes over from osmotic control as dehydration proceeds, this results from the stimulation of anti-diuretic hormone secretion by the reduced plasma volume.

These, however, are theoretical aspects which do not affect the value of the book as a practical manual.

A Practical Handbook of Midwifery and Gynæcology: For Students and Practitioners. By W. F. T. Haultain, O.B.E., M.C., B.A., M.B., B.Ch., F.R.C.P.Ed., F.R.C.S.Ed., F.R.C.O.G., and Clifford Kennedy, M.B., Ch.B., F.R.C.S.Ed., F.R.C.O.G., including a section on the "Management of the Infant and Neo-Natal Conditions", by J. L. Henderson, M.D., F.R.C.P.Ed.; Fifth Edition; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. [illegible], with 48 illustrations. Price: **30s.**

IN the preface to the fifth edition of their handbook, the authors express the hope that adequate information in obstetrics and gynæcology can be obtained readily and quickly from its pages.

There is a tremendous amount of useful information in the text, the style is concise, and much of the work is presented in tabulated form. The handbook commences with a short study of anatomy and physiology, and proceeds through the course of normal and abnormal pregnancy and labour to a discussion of gynæcological problems, and finally to a chapter on infant care.

The influence of domiciliary midwifery, as opposed to the greater incidence of hospital deliveries in this country, is apparent in the section dealing with the management of normal labour and elsewhere. Many practices—for example, the use of sodium bicarbonate in the treatment of heartburn of pregnancy and the application of forceps to the breech—are at variance with local teaching. In the chapter on puerperal sepsis, no mention is made of any antibiotic other than penicillin.

The student or practitioner will find much of use in this volume, but will need to be well aware of the essential differences between some of the methods advocated and the teaching in this country.

The Diagnosis and Treatment of Pulmonary Tuberculosis. By Paul Dufault, M.D., with a chapter on pathology by A. Reynolds Crane, M.D., and a chapter on pulmonary function by Oscar Feinsilver, M.D.; Second Edition; 1957. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 7¼" x 5¼", pp. 428, with 162 illustrations. Price: 99s.

"It is hoped that all interested in the medical and social aspects of Tuberculosis will find concise and practical information in the following chapters. We did not try to be exhaustive, but we hope to have been complete." So writes Dr. P. Dufault in the preface to this excellent little book.

It must be confessed that our initial reaction on receiving this book for review was "not another book on tuberculosis?"; but the further we delve into the book, the more we realize the truth of Dr. Dufault's statement in his preface. By its very nature this is not a book for the specialist in tuberculosis, but more for the practising physician who wishes to keep in touch with what his colleagues are doing in this field of medicine. It certainly covers all aspects of tuberculosis today and perhaps a little of yesterday, for quite a large section is devoted to artificial pneumothorax, a procedure which has lost favour with almost all physicians in the western world.

A very good section devoted to respiratory physiology and the place of lung function tests today has been contributed by Dr. O. Feinsilver, and this alone makes the book worth reading.

The Year Book of Dermatology and Syphilology (1956-1957 Year Book Series). Edited by Rudolf L. Baer, M.D., and Victor H. Witten, M.D.; 1957. Chicago: The Year Book Publishers, Incorporated. 7¾" x 5", pp. 464, with 62 illustrations. Price: $7.00.

IN their introduction to this Year Book the editors state that in the clinical appraisal of therapy with modern drugs "interest has centered particularly on the effects of long term administration of the corticosteroids, antibiotics, antimalarials and other systematically given drugs". After first condemning the systematic use of novobiocin, they report favourably on the long-term use of the tetracyclines, noting that drug eruptions are remarkably few, apart from monilial complications. They note extensive clinical trials of antimalarial drugs, record the search for similar compounds with greater therapeutic effect but lower toxicity and sensitizing capacity, and refer to investigations of the mechanisms of these drugs. Tranquillizing drugs appear to have disappointed the hopes first placed in them for

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help in the management of various dermatoses. They mention increasing interest in soft quality X rays, which is likely to be accelerated by the current concern over the long-term effects of the more penetrating forms of radiation.

Important experiments in the artificial transfer of infectious skin diseases to human volunteers are reviewed. A group of Spanish workers conducted extensive experiments with the transfer by inoculation of onychomycosis due to various organisms, which the editors hail as "probably the most daring and certainly one of the most interesting and informative pieces of work in this year's dermatologic literature". Baer and co-workers investigated the experimental transfer of dermatophytosis of feet; their findings have interesting public health implications. Another group did a series of intracutaneous inoculations of syphilis into 62 human volunteers from Sing Sing Prison, with results which, according to the editors, answer "some of the many important questions about which there has been much speculation in the past".

This book begins with a comprehensive chapter on allergic eczematous contact dermatitis, subtitled "A Review of Selected Aspects for the Practitioner". The remainder of the volume is composed of abstracts of articles published from December, 1955, to November, 1956, arranged under the same headings as in the preceding volume.

Hale-White's Materia Medica, Pharmacology and Therapeutics. By A. H. Douthwaite, M.D., F.R.C.P. (Lond.); Thirtieth Edition; 1957. London: J. and A. Churchill, Limited. Price: 24s.

THIS new edition of a well-known text-book will be welcomed, as so many new drugs have appeared since the last edition was published five years ago, and so much more is known about the drugs that were new then. This work has been completely revised, and the editor is to be congratulated on producing an up-to-date volume which can serve as a handy book of reference on all aspects of the subject, and contains useful summaries on all the newer drugs. The section "Endocrine Preparations and Sex Glands" was revised by Dr. P. M. F. Bishop, and Dr. Roy Goulding, Lecturer in Pharmacology at Guy's Hospital Medical School, assisted in the general revision.

Atlas of Tumor Pathology (Washington: Armed Forces Institute of Pathology). Section 8—Fascicle 30: "Tumors of the Kidney, Renal Pelvis and Ureter", by Balduin Lucké, M.D., Dr.P.H., and Hans G. Schlumberger, M.D.; 1957. 10¼" × 7⅝", pp. 208, with 199 illustrations. Price: $2.25. Section 10—Fascicle 36: "Tumors of the Pituitary Gland and Infundibulum", by James W. Kernohan, M.D., and George P. Sayre, M.D.; 1956. 10¼" × 7⅝", pp. 84, with 67 illustrations. Price: $1.00. Section 10—Fascicle 38: "Tumors of the Eye and Adnexa", by Algernon B. Reese, M.D.; 1956. 10¼" × 7⅝", pp. 208, with 122 illustrations. Price: $2.00.

THESE fascicles are part of a comprehensive series issued by the Armed Forces Institute of Pathology of the United States of America, to which we have made a number of previous references. They contain magnificent reproductions of the macroscopic and microscopic appearance of tumours of the pituitary gland, of the eye and adnexa and of the urinary system. Some are in colour, some are in black and white. Each section begins with a description of the neoplasms of the region concerned relating to their natural history, histogenesis and appearance.

Tumours of the pituitary gland and infundibulum are dealt with in Fascicle 36 of Section 10 by James Kernohan and George Sayre. The fascicle begins with a description of the normal anatomy and structure of the gland. Benign and malignant tumours of the gland are considered in turn, and there is a section on hypopituitarism not associated with primary tumours of the gland, such as that due to pressure from an overlying meningioma or carcinoma.

In the fascicle on the eye, Fascicle 38 of Section 10, benign and malignant growths of mesodermal and neuroectodermal epithelial origin are described, with a special chapter on melanomata. Tumours of the lachrymal gland are also dealt with, as are metastatic tumours and orbital neoplasms.

In Fascicle 30 of Section 8 are tumours of the kidney, renal pelvis and ureter. They are classified according to their cell of origin and their site. Precancerous conditions such as leucoplakia of the renal pelvis are illustrated, as is renal cholesteatoma.

The fascicles provide an excellent guide to the pathology of the variety of neoplasms affecting the organs listed. The printing, especially of the illustrations, is beyond reproach. Each page is loose-leaf, so that the illustrations can be projected onto a screen for teaching purposes.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Expert Committee on Insecticides: Seventh Report"; World Health Organization Technical Report Series No. 125; 1957. Geneva: World Health Organization. 9¼" × 6½", pp. 32. Price: 1s. 9d.

This report is mainly concerned with resistance of insects to insecticides. A short section deals with "disinsectization" of aircraft.

"Modern Medicine for Nurses", by Patria Asher, M.D., M.R.C.P., with a chapter on mental ill health by Portia Holman, M.D., M.R.C.P., D.P.M.; Fourth Edition; 1957. London, Toronto, Melbourne, Cape Town: William Heinemann (Medical Books), Limited. 8½" × 5¼", pp. 392, with 99 illustrations. Price: 22s. 6d.

Revised and enlarged since the publication of the previous edition in 1954.

"Functional Neuro-Anatomy: Including an Atlas of the Brain Stem", by A. R. Buchanan, M.D.; Third Edition; 1957. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10" × 7", pp. 364, with 273 illustrations. Price: 82s. 6d.

Extensively revised since the publication of the previous edition in 1951.

"Disorders of the Blood: Diagnosis, Pathology, Treatment, Technique", by Sir Lionel E. H. Whitby, C.V.O., M.C., M.A., M.D. (Cantab.), F.R.C.P. (Lond.), D.P.H., and C. J. C. Britton, M.D. (New Zealand), D.P.H.; Eighth Edition; 1957. London: J. and A. Churchill, Limited. 9½" × 6", pp. 888, with 145 illustrations. Price: 75s. (English).

The previous edition was published in 1953. The extensive revision for the present edition was completed before the death of Sir Lionel Whitby.

"Chemical Methods in Clinical Medicine: Their Application and Interpretation with Techniques of Simple Tests", by G. A. Harrison, B.A., M.D., B.Ch. (Cantab.), M.R.C.S. (Eng.), F.R.C.P. (Lond.), F.R.I.C.; Fourth Edition; 1957. London: J. and A. Churchill, Limited. 9" × 6", pp. 680, with 163 illustrations. Price: 65s. (English).

The previous edition was published in 1947. The book has been fully revised.

"The Closed Treatment of Common Fractures", by John Charnley, B.Sc., M.B., F.R.C.S.; Second Edition; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 9½" × 7", pp. 272, with 199 illustrations. Price: 50s. (English).

New material has been added since the publication of the first edition in 1950.

"Progress in Neurobiology: 2. Ultrastructure and Cellular Chemistry of Neural Tissue", edited by Heinrich Waelsch, M.D., with 37 participants; series editors, Saul R. Korey, M.D., and John I. Nurnberger, M.D.; 1957. New York: Paul B. Hoeber. 9¼" × 6", pp. 272, with 40 illustrations. Price: $7.50.

A symposium sponsored by the American Neurological Society and the Medical School of Western Reserve University.

"Dermatologic Formulary: From the New York Skin Cancer Unit, Service of Dermatology (Dr. Marion B. Sulzberger, Director)", edited by Frances Pascher, M.D.; revised 1957. New York: Paul B. Hoeber. 7¼" × 5", pp. 191. Price: $4.00.

Completely revised since the publication of the first edition in 1953.

"Pneumo-encephalography", by E. Graeme Robertson, M.D. (Melb.), F.R.C.P., F.R.A.C.P.; 1957. Oxford: Blackwell Scientific Publications. 10" × 7½", pp. 408, with 209 illustrations. Price: £5 10s.

A new book based on first-hand experience and dealing with the anatomical, physical and clinical aspects of pneumoencephalography.

"Gastro-Duodenal Ulcer: Physio-Pathology, Pathogenesis and Treatment", by J.-Jacques Spira; 1956. London: Butterworth and Company (Publishers), Limited. 9¾" × 6¾", pp. 568, with several illustrations. Price: £5.

The author describes this as "a fresh approach to the problem and an attempt to clarify the many paradoxes which confuse the student of gastro-enterology".

The Medical Journal of Australia

SATURDAY, DECEMBER 14, 1957.

CARBUTAMIDE AND TOLBUTAMIDE IN THE TREATMENT OF DIABETES.

IT is often to be regretted that the use of new drugs for the treatment of specific diseases is discussed in the public Press while it is still very much in the experimental stage. A recent example is the use of tolbutamide in the treatment of diabetes. A paragraph about it appeared in a newspaper which, although conservatively worded, so that it did not suggest too much, inspired a question in the Senate as to placing the drug on the free list. This suggestion was at best premature, and indeed many competent people regret the fact that drugs for the oral treatment of diabetes have been released to the public at all, even though on a doctor's prescription. However, since they have been released, it is important to know their uses and their limitations.

In 1955 certain German investigators noted that the administration of some sulphonamides caused a reduction in the level of blood sugar. One of these, 1-butyl-3-para-amino-benzene sulphonylurea (BZ55), later called carbutamide, was considered worthy of extensive clinical investigation. Two groups of German workers, H. Franke and J. Fuchs, and F. Bertram, E. Benfeldt and H. Otto in October, 1955, in the *Deutsche medizinische Wochenschrift*, published the results of clinical trials with carbutamide. Reduction in blood sugar level and control of glycosuria were observed in 80% of diabetic patients receiving the drug. Later observations made in other parts of the world were published, and these have been summarized by R. H. Unger and J. W. Davidson, junior.[1] Bertram and his co-workers found that most mildly diabetic subjects over the age of 59 years reacted well. None of them had been using insulin. Another similar group receiving insulin were controlled satisfactorily when carbutamide replaced insulin. In younger patients carbutamide therapy did not significantly alter insulin requirements. Other observers obtained similar results. E. Downie, J. Bornstein and B. Hudson[2] in Melbourne made clinical trials with carbutamide on a selected group of 28 patients, none of whom were juveniles. There were 19 successes and seven failures. Five of the patients responding to carbutamide had previously required insulin for control.

The general opinions of workers who have investigated the use of carbutamide may be summarized as follows. Carbutamide is effective in the management of diabetes of the "adult" type, characterized by mildness and stability of the disease, a relative tolerance to insulin, maturity at onset and often obesity. Failure to respond to carbutamide can be expected in the "juvenile" type, characterized by severity and lability of the disease, insulin sensitivity and onset during youth. Side reactions are important. Earlier workers found no serious side reactions following the use of carbutamide. However, Downie *et alii* observed four cases of granulocytopenia in their 26 patients, although it was not extreme or persistent. J. C. Phemister[1] and J. D. Baird and L. J. P. Duncan[2] have reported the occurrence of thrombocytopenia and leucopenia in several patients treated with carbutamide. They suggest that the toxic potentialities of this drug preclude its clinical use in the future, and that further long-term clinical studies should be confined to tolbutamide. Other reports confirm the dangers associated with the use of carbutamide, and apparently it has been withdrawn from distribution, in America at least.

Shortly after the introduction of carbutamide another compound was made available which differed from carbutamide only in having the NH_2 group attached to the benzene ring replaced by a CH_3 group. The substance is 1-butyl-3-p-toluene sulphonylurea and is called tolbutamide. Carbutamide is a sulphonamide and, in addition to its hypoglycæmic properties, has a potent antibacterial action. Tolbutamide has no antibacterial action, but acts in almost the same way as carbutamide in producing hypoglycæmia. The only side effects which seem to have been noted in the use of tolbutamide are occasional skin rashes.

Unger and Davidson treated 18 diabetics with tolbutamide. Seven of 12 patients with stable adult diabetes given three to six grammes per day showed diminution in fasting hyperglycæmia and suppression of glycosuria. All but one of these were adequately controlled by diet alone. Large doses of tolbutamide did not benefit patients with labile diabetes. Baird and Duncan have analysed the hypoglycæmic response to tolbutamide in contrast to that to carbutamide. It has been assumed that the response to and hence the therapeutic administration of the two drugs are identical. This is not the case. The hypoglycæmic response to a single dose of tolbutamide is of much shorter duration than that obtained with carbutamide, owing to the difference in the rate of fall in the concentration of the two drugs in the blood. Both drugs are rapidly absorbed, but there is considerable variation in the plasma level of both drugs following the administration of the same dose. Carbutamide escapes in the urine relatively slowly, the "half life" in the blood being 30 to 60 hours. The half life of tolbutamide on the other hand is about five to eight hours; so that if single doses of 1·5 grammes are given daily, in most cases eight to ten hours after the administration of the drug the concentration in the blood has fallen below the minimum effective range. On the other hand, the effect of carbutamide is cumulative. Because of this most patients will achieve adequate therapeutic levels throughout the 24 hours by taking the drug twice a day, the first dose at breakfast time and the second eight to ten hours later. Tolbutamide does not improve glucose tolerance, and its hypoglycæmic effect is due solely to a

[1] *J.A.M.A.*, 1956, 162: 447 (September 29).
[2] *Australasian Ann. Med.*, 1957, 6: 105 (May).

[1] *Brit. M. J.*, 1957, 1: 199 (January 26).
[2] *Scottish M. J.*, 1957, 2: 341 (September).

reduction in the fasting glucose level. Diabetic patients most suitable for treatment with tolbutamide are those who show moderately high fasting glucose values in the blood with relatively good glucose tolerance. Certain patients, in middle or later life, who have recently developed diabetes may be well controlled by one dose per day.

Both drugs appear to have the same effect on glucose metabolism. How they act is not yet known with certainty. They are in no way insulin substitutes. The liver is essential for their action, and there is considerable evidence that they act on the process of neogenesis of glucose from proteins in the liver. Downie and his colleagues state: "It is a sobering thought to realize that the absence of glycosuria in a patient treated with a sulphonylurea may prove to be a measure of harmful inhibition of the metabolism of protein by the liver."

To sum up, carbutamide can be ruled out because of toxic effects, but it would seem that in certain cases of diabetes tolbutamide may be a useful drug. An important comment is made by P. K. Bondy in an editorial note in the section on metabolism of "The Year Book of Medicine (1957-1958 Series)" at page 692: "There is no evidence that tolbutamide is a substitute for insulin; indeed its effects are quite different from those of the hormone. The critical question is whether reducing the blood sugar without appreciably altering the rate of glucose utilization is important. Certainly the basic principles of good diabetic management still apply: adequate dietary control, insulin if needed, prevention of obesity are all important. In a few cases, tolbutamide may prove to offer something of value to patients who are blind or otherwise find insulin hard to administer; but for most diabetics the new drugs still appear experimental." There is yet much to be learned of the activity even of tolbutamide before its general therapeutic use can be recommended.

Current Comment.

MATERNAL MORTALITY.

THE spectacular decrease in maternal mortality over the past 25 years is common knowledge, but a recent report published by the British Ministry of Health[1] brings the subject up to date with a wealth of fascinating detail and authoritative comment. This report may profitably be read in conjunction with that of the Victorian Consultative Council on Maternal Mortality.[2] The two reports differ considerably in scope. The Victorian report (read at a meeting of the Victorian Branch of the British Medical Association) is an analysis of all recorded deaths "in pregnant women from any cause whatsoever, from the time of conception up till 28 days after (delivery)" in the State of Victoria in the three years 1954 to 1956. It deals with 117 cases of maternal death and attempts to allocate them as "avoidable" or "unavoidable". The British Report analyses confidential reports on 1094 deaths classed as due to pregnancy and childbirth, and 316 due to associated causes (principally heart disease), occurring in the three-year period 1952 to 1954. These reports comprise 71% of all registered maternal deaths, and are considered to be a fully representative sample.

The authors of the British report use the concept of the "primary avoidable factor", adding that "there can be no certainty that all these deaths could have been actually prevented, but in all of them the risk of death could have been materially lessened". This appears not merely diplomatic, but a more truthful way of stating the actual position than a blunt classification into avoidable and unavoidable deaths. An avoidable factor was considered to be present in 45·9% of the cases investigated. In nearly one-quarter of these cases the avoidable factor was the patients' refusal or neglect to follow medical advice, or to seek such advice, but the report adds that "many probably rated their home responsibilities above the care of their own health" and suggests that the remedy lay in better follow-up services by hospitals and clinics. The other main avoidable factors were inadequate ante-natal care, failure to make use of the "flying squad" services, and unwise arrangements for confinement at home or in small maternity homes of patients with a high obstetric risk. These findings recall important differences between British and Australian practice. In England and Wales one-third of all confinements take place in the patient's home, and "flying squad" services are generally available. However, the report points out that an estimated total of 570 maternal deaths with an "avoidable factor" occurred in just over two million confinements—that is, roughly one in 4000 deliveries. (This calculation omits deaths following abortion and ectopic pregnancies, as these are outside the scope of the maternity services.) Room for improvement is therefore small, though these figures are not regarded with complacency.

The main causes of maternal death are grouped as: (i) toxæmia (including toxæmic accidental hæmorrhage), 22%, (ii) hæmorrhage (excluding toxæmic accidental hæmorrhage), 17%, (iii) abortion, 14%, (iv) pulmonary embolism, 13%. Important sections are devoted to each of these causes, as well as to abortion, cardiac disease associated with pregnancy, Cæsarean section and deaths due to complications of anæsthesia. An interesting calculation is an estimated mortality of four per 1000 cases of delivery by Cæsarean section for the country as a whole. The dangers of obstetric anæsthesia are stressed, the commonest cause of death in this category being inhalation of stomach contents (32 out of 49 deaths), and it is suggested that the management of such cases "should not be undertaken by unsupervised and inexperienced junior medical staff". The point is emphasized that "the single-handed management of anæsthesia and delivery is excusable only in extreme emergency where no help is available".

In conclusion, it may be of interest to quote some figures of maternal mortality given in a recent WHO Press release (all figures are per 1000 live births): United States, 0·3 for the white population and 0·5 for the population as a whole; New Zealand, 0·4; Scotland, 0·5; Australia, 0·6; England, 0·7.

WHO "DISCOVERED" THE BLOOD CIRCULATION?

A CONTROVERSIAL AND ENTERTAINING PAMPHLET has been received from Alcmaeon Publications in New York.[1] After careful examination it was found to be completely innocuous and to consist of "some authoritative opinions and judgements expressed by renowned international scholars on the discovery of the blood circulation made by Cesalpino of Arezzo in 1571". It would seem that another subtle attempt is being made to open up an old wound and to convert into an international issue the futile argument as to which country is entitled to the honour and glory of priority for the discovery of the blood circulation. In a lengthy introduction the publishers eloquently define the main points in support of a case for the plaintiffs, while the

[1] "Report on Confidential Enquiries into Maternal Deaths in England and Wales, 1952-54", Reports on Public Health and Medical Subjects, No. 97. 1957: Her Majesty's Stationery Office, London.

[2] M. J. AUSTRALIA, 1957, 2: 334 (August 31).

[1] "Consideration About Cesalpinus' and Harvey's Works on the Blood Circulation Discovery"; 1957. New York: Alcmaeon Publications. 10½" × 8", pp. 27. No price stated.

rest of the pamphlet is taken up with a number of extracts from the writings of various medical authors expressing regrets and polite resentment that "the dogma which affirms the sacred supremacy of William Harvey" should be so rigidly frozen into the minds of Anglo-Saxon and American peoples. Recent reports indicate that is just as firmly frozen into the minds of the Russian peoples.

The present discussion has arisen out of the publication of a book written in 1945 by Dr. G. P. Arcieri, a professor of medical history at Rome, in which he endeavoured somewhat impassionately to sustain the claims of his compatriots, Realdo Colomo and Andrea Cesalpino, to general recognition as the true discoverers of the pulmonary and systemic circuits. There can be no doubt that Arcieri wrote his book in all good faith, and that it was, in truth, a serious and accurate study of the whole problem. In any case, it has stimulated renewed interest in many academic quarters, and has induced scholarly physicians to make a fresh appraisal of the recorded statements, experimental work and relative merits of Galen, Ibn-an-Nafis, Servetus, Colombo, Cesalpino and Harvey in arriving at the scientific truths essential for a better understanding of the circulation of the blood.

When all is said and done, one undeniable fact emerges from the uncertainties and vague circumstances of a long past: with the exception of Claudius Galen, who came from Asia Minor, and William Harvey, the typical Englishman, the views of the other contestants had no practical effect on the current medical thought of their own time; and their unsupported theories received little or no attention from later generations of anatomists once the experimental method of research became firmly established as a safe and sure pathway to the acquisition of new knowledge.

Today we can be quietly amused at the remark of one American contributor to the pamphlet that "the Harvey press agents have been most active", and at the good-natured jibe of another who writes that "one begins to realize again why the Latins speak of 'perfidious Albion' and of 'Britannia waives the rules'". But once again let us invoke the spirit of Sir William Osler to plead the cause of defendants in the case, as he did so eloquently in similar circumstances nearly forty years ago:

> These men with others doubtless, were in latent possession of the truth. But every one of them saw darkly through Galenical glasses, and theirs was the hard but common lot never to reach such conscious possession as everywhere to make men acquiesce. One must have the disinterestedness of the dead to deal with a problem about which controversy has raged, and in which national issues have been allowed to blur the brightness of an image which would be clear as day to those with eyes to see. Nor would I refer to a matter long since settled by those best competent to judge, had not the well known work of Luciani, the distinguished Professor of Physiology at Rome, appeared recently in German dress, edited by Professor Verworn, and spread broadcast views to which, with a chauvinism unworthy of their history, our Italian bretheren still adhere. It has been well said "that he alone discovers who proves", and in the matter of the circulation of the blood, this was reserved for the pupil of Fabricius.

THE HEALTH OF BRITAIN.

THOUGH the subject matter of the latest annual report of the British Ministry of Health[1] is now about two years old, it contains much that is of interest on a wide variety of topics. We are all aware that the mortality from most infectious diseases has been diminishing rapidly, but the details of what has been achieved and what remains to be done still make absorbing reading.

At the end of 1955 there had been no case of smallpox in Britain for two years, though this immunity was soon to be interrupted. Vaccination is no longer compulsory in Britain, but is officially encouraged, and a little over one-third of all children under the age of one year were in fact vaccinated. The vaccination rate, however, varies enormously in different parts of the country, being as little as 3% in one county borough and as high as 70% in another. This probably reflects in part the amount of attention given to publicity by different health authorities. That this procedure is not entirely without risk is shown by the notification of two deaths out of over 300,000 persons receiving primary vaccination, one of these was in a case of generalized vaccinia in an infant believed to be suffering from agammaglobulinæmia; the other was in one of six reported cases of post-vaccinal encephalomyelitis.

Only 155 cases of diphtheria were notified, the lowest annual figure so far recorded, but 71 of these occurred in five distinct outbreaks, indicating that in some communities the proportion of non-immunized persons was still high enough to permit an outbreak to gain momentum. Two of these outbreaks were in schools for educationally subnormal children, and it is suggested that this is connected with the difficulty in obtaining parental consent for immunizing procedures in many such cases.

The incidence of scarlet fever continues to decline. In the year under review only two deaths were recorded in the acute stage of this illness; in 1870, 32,500 deaths were attributed to scarlet fever. There was a further remarkable drop in the incidence of whooping-cough (climatically, 1955 was a very favourable year from the public health point of view), and the case fatality ratio was the lowest on record, most of the few deaths being in infants under the age of one year. Notifications of poliomyelitis showed a sharp up-turn, 3712 paralytic cases having been recorded; the highest incidence was in the first ten years of life, but mortality from this disease was highest in the 25 to 34 years age group.

A separate chapter is devoted to discussion of the question of vaccination against poliomyelitis, but this is now mainly of historical interest. The 1955 outbreak in England included a very high proportion of non-paralytic cases, and there was considerable difficulty in deciding to what extent these might be due to other neurotropic infections. At least one such outbreak was identified, and the report concludes that "there can be little doubt that benign neurotropic infections other than, but similar to non-paralytic poliomyelitis, occur in Britain, particularly during the summer months".

For the first time since 1947 the progressive fall in notifications of syphilis received a slight setback, but new cases of infantile congenital syphilis were at a record low level of 41 cases with no fatalities. A glance back to 1917 shows that the infant mortality from congenital syphilis alone then reached the appalling figure of two per 1000 live births. The incidence of gonorrhœa also showed a slight increase, and there has been no appreciable improvement in the statistics of this disease over the past five years. In males, the number of cases of non-gonococcal urethritis has been progressively increasing since this condition was first made notifiable in 1951, and now approximately equals the figure for gonorrhœa.

The chapter on laboratory services covers a variety of topics. It is stated that over the past few years food poisoning has continued to become more and more prevalent, and this is attributed to the continuing increase in communal feeding—"the home was being gradually displaced by restaurants and canteens". For some years past occasional outbreaks of paratyphoid B fever had been traced to synthetic cream used in bakery products, but there the trail ended till, in the year under review, two such outbreaks were traced to bakeries using Chinese frozen eggs, from which bacteria of the incriminated strain were isolated. Further investigation showed that Chinese frozen eggs were commonly contaminated with organisms of the salmonella group, especially *Salmonella thompson*. It is believed that synthetic cream may become infected from contaminated mixing machines. In recent years *Salmonella thompson* has been the third commonest cause of food poisoning in England and Wales; the source of this organism had previously been a mystery, as it is otherwise seldom found in Britain.

[1] "Report of the Ministry of Health for the Year Ending 31st December, 1955. Part II: On the State of the Public Health", Her Majesty's Stationery Office, London.

Other chapters deal with a wide variety of subjects as they affect the health of the community, and contain much of general interest. The facts mentioned in this article are only a small sample of the information to be found in this very readable report, and few who take the trouble to peruse it will regret the time so spent.

CANCER OF THE UTERINE CERVIX.

O. G. A. BERGGREN, of the Gynæcological Department of the Radiumhemmet, Stockholm, notes that Sweden is a particularly appropriate country in which to study the social and hygienic factors underlying the incidence of cancer of the uterine cervix;[1] for in Sweden, since 1928, practically every patient with cancer of the cervix has been treated at one of the four "Jubilee Clinics" by radium implantation. Berggren has concentrated his attention on morbidity—that is, case incidence—especially in the years of the five-yearly Swedish census. He rather surprisingly omits any reference to death rates from the disease. In his Table I, he gives case incidence rates from A. Lindell's[2] study, on which we[3] have already commented. There was an increase in the incidence rates of cervical cancer of perhaps one-third between the years 1930 and 1947, and Berggren proposes to find the cause.

Berggren's second chapter considers the possible causes of cancer of the uterine cervix, and here it is evident that the role and importance of the various ætiological factors are still very much in doubt. On the racial side, it is known that cancer of the cervix is uncommon in Jewesses; but only a few authors believe that this is truly genetic and hereditary. The influence of parity is much stressed by some authors, and Berggren's Table II from H. L. Kottmeier,[4] also of the Radiumhemmet, gives the parity of patients. Unfortunately no control data are given, so that proper comparisons cannot be made. In Australia, we may note, H. O. Lancaster[5] has analysed the official statistics and finds that married women suffer from more uterine cancer than unmarried women, but that married women who have borne children do not suffer more than married women who have not borne children. There is a good deal of evidence that sexual relations rather than child-bearing are of importance. Thus F. Gagnon[6] reported no cases of cervical cancer, but 12 cases of cancer of the body of the uterus, in a study of 13,000 nuns. E. L. Kennaway[7] believes abstinence from intercourse to be of importance in producing the relative immunity of Jewish women. The great preponderance of cervical cancer in married women is well known, but it is also unduly common in the divorced and in prostitutes. Wynder, Cornfield, Schroff and Doraiswami have further shown that cervical cancer is common in those who marry early and in those who marry twice. Kennaway[7] has shown that there is a distinct social gradient in the mortality rates in England and Wales. We conclude our consideration of this chapter of Berggren's monograph with the impression that hygienic factors are very important.

Berggren's third chapter gives a review of the evidence for the observation that in Denmark there are higher rates for cervical cancer in the cities than in the country, and his sixth chapter analyses similar Swedish data. There is very good evidence that the morbidity (by which Berggren evidently means case incidence during a period rather than prevalance at a point of time) is higher in Stockholm than in other parts of Sweden. We have the impression that this discussion would have been strengthened by a study of the experience of three or even five years about a census rather than of a single year, since when Sweden is subdivided into 28 counties, the numbers of uterine cancers to be expected in some is quite small if only a single year is considered.

In Chapter IX, Berggren discusses stage. Here it may have been appropriate to add a little on mortality statistics. There must remain also some doubt that the increased incidence over the fifteen-year period from 1935 to 1950 may have been due to increased interest in and search for the disease. This seems to be borne out by the increase in the proportion of Stage I cancers in Stockholm—for example, from 7% in 1935 to 26% in 1950.

In conclusion, it may be noted that the author has given a very readable account, including a valuable bibliography, of the demographic features of cancer of the uterine cervix, and the production is excellent.

[1] "Demographic Studies on Carcinoma of the Uterine Cervix in Sweden", by O. G. A. Berggren; Part I; 1957. Stockholm: Acta Radiologica Supplement 145. 9¾" × 7¼", pp. 148. Price: 30 Sw. Kr.

[2] "Carcinoma of the Uterine Cervix; Incidence and Influence of Age", *Acta radiol.*, 1952, *Supplementum* 92.

[3] M. J. AUSTRALIA, 1952, 2:445 (September 27).

[4] "Carcinoma of the Female Genitalia", Abraham Flexner Lectures, 1953, Williams & Wilkins, Baltimore.

[5] M. J. AUSTRALIA, 1951, 2:1 (July 7).

[6] *Am. J. Obst. & Gynec.*, 1950, 60:516 (September).

[7] *Brit. J. Cancer*, 1948, 2:177 (September).

INSECT RESISTANCE.

THE introduction of DDT during the war years heralded a new era in insect control, and for a time it seemed that the way was open to the complete control of at least some of the important disease vectors. Few people then guessed that resistant strains would soon appear to complicate the picture. Since then the introduction of new and improved insecticides has evoked the emergence of insect strains resistant to each in turn, and it has become apparent that poison-resistant strains in insects pose a major problem comparable with that of antibiotic-resistant strains in bacteria. This problem forms the main subject matter of a recent report from the World Health Organization.[1] Through the use of modern insecticides great advances have been made in the control of many insect-borne diseases, and the question of resistant strains is one of the main impediments to further progress. Stating that "the problem of resistance is growing much more rapidly than are measures to deal with it", the members of the Committee in their report call for greatly increased effort in research into this problem, which they regard as at present quite inadequate. In proportion to the amount of money being spent on insect control, that devoted to research on this basic problem is "inadequate and out of line with sound economic practice". They also note the scarcity of highly qualified research workers available for such work. They then proceed to outline their suggestions for a programme of research, varying from investigations into the present status of resistance (resistant strains are known in at least ten major disease-vector species) to such fundamental questions as the physiology and genetics of resistance. The resistance of the house-fly to DDT has been extensively investigated, and it has been found that "enzyme degradation" of the insecticide is a major factor, though probably not the only one. In a final section on alternative control measures where resistance has developed, mention is made of an intriguing success. Effective control of a blowfly, *Callitroga hominivorax*, on the island of Curaçao was obtained by liberating large numbers of males previously sterilized by irradiation.

[1] "Expert Committee on Insecticides: Seventh Report"; World Health Organization Technical Report Series No. 125; 1957. Geneva: World Health Organization. 9¾" × 6¼", pp. 32. Price: 1s. 9d.

BIOLOGICAL ASPECTS OF THE TRANSMISSION OF DISEASE.

THE tools and techniques employed by workers investigating problems in the branches of biological science are as many and varied as the resources of human ingenity can devise. The constant interchange of facts, principles and methods is as important to each one of them as his own most cunningly devised experiment of the moment. Those disciplines which seek to study change in the living animal in competition with his neighbours, his environment or his nutriment, the process which engenders the reaction we call disease, are especially in need of the results of observation on animals whose natural term of life is short enough for man to record and analyse. The

Institute of Biology serves this need admirably by the publication of the proceedings of its symposia, together with informed discussion upon the papers. A volume entitled "Biological Aspects of the Transmission of Disease", edited by C. Horton Smith, is the latest of these.[1] The scope of subjects is spread over the range of human, animal and plant pathogens in 22 fairly short papers, the whole volume covering 179 pages. It would be of little value to enumerate their titles here, and a single reviewer is hopelessly unable to criticize their content adequately. Therefore, he must needs pay attention to his knowledge of the authors, and the assembly of names under the list of contributors offers ample proof of the respect which must be paid to this book. Suffice it to say that views on the transmission of disease between plants, the reservoir function of the soil for pathogenic microorganisms and the veterinary problems of the health of the animals make an array which covers an enormous portion of the biological spectrum.

Special mention may be made of the thought-provoking essay of C. A. Andrewes. In this aspect of modern science, the senior man with years of practical work, experience and observation accumulating in his mind seeks broader horizons both in time past and in time to come. It is true of Andrewes, equally so of his colleague Burnet; the evolutionary developments leading to the present problems have a special fascination for them both. Either could have voiced the closing sentence of Andrewes's essay: "While the pageant of metazoon evolution moves so slowly that we can hardly detect its progress, virus diseases seem to be appearing, waxing and waning within the lifetimes of human observers." This book should find a place amongst the reference libraries of every biological laboratory in the country.

GLYCYRRHITINIC ACID AS AN ANTI-INFLAMMATORY AGENT IN DERMATOLOGY.

In the "Current Comment" columns of this Journal of August 15, 1953, and of May 5, 1956, attention was drawn to papers dealing with the action of liquorice and of its active principle, glycyrrhitinic acid, in causing retention of water, sodium and chloride in man and to its cortisone-like activity. It was inevitable that sooner or later somebody would investigate the action of glycyrrhitinic acid as an anti-inflammatory agent in skin diseases in view of the pronounced effect hydrocortisone has in this regard. This has now been done by E. Colin-Jones and G. F. Somers.[2] They have found that active fractions of crude glycyrrhitinic acid are active anti-inflammatory agents both when applied locally and when injected systemically. Artificially induced inflammatory changes in animal skins were quickly inhibited when ointments containing the acid were applied locally. Systemic administration of glycyrrhitinic acid suppressed both acute and chronic swelling in the paw of a rat following the injection of a 3% solution of formaldehyde into the joint.

Ointments, greasy and non-greasy, were prepared containing 2% of the isomers of glycyrrhitinic acid shown to be biologically active and used in the treatment of skin diseases in man. Dramatic results were often obtained in acute inflammatory conditions such as contact dermatoses, but the results in chronic diseases were much less convincing, or there was no reaction at all. A comparison between hydrocortisone ointment and the glycyrrhitinic acid ointment showed very similar results in acute conditions. In 254 cases of a variety of skin diseases the results given by glycyrrhitinic acid ointment indicated anti-inflammatory action. Careful controls seem to have been used to eliminate psychosomatic factors in the patient. In general the effects were very similar to those obtained with hydrocortisone ointments, but sometimes one succeeded when the other failed and vice versa. In some cases better results were obtained by using an antibiotic in the ointment as well as glycyrrhitinic acid.

If the results recorded by Colin-Jones and Somers are confirmed, the ointment may be very useful in cases in which it is considered inadvisable to use hydrocortisone.

LISTERIOSIS.

Listeriosis has long been regarded as a rare disease, but it appears that it is being recognized with increasing frequency. It was first described in guinea-pigs and rabbits in 1926, while the first human case was noted in 1929. The causal organism, *Listeria monocytogenes*, is a small, non-sporing, Gram-positive rod, which is motile. It grows readily in nutrient media. M. Hood[1] has given a detailed account of the disease based on ten cases observed in the Charity Hospital of Louisiana in New Orleans. Four other cases were known to have occurred in Louisiana in the two years of the investigation. Hood's account makes it clear that the disease is very varied in its manifestations. Six types have been described: (i) the septic type with mononucleosis, (ii) the oculo-glandular form, (iii) the septic-typhoid form, (iv) infection of the central nervous system, (v) granulomatous sepsis of the newborn, (vi) listeriosis of pregnancy. Adequate blood culture will determine the presence of the septic-typhoid form of listeriosis. Infection of the central nervous system produces dramatic effects and is the commonest type. Examination of the spinal fluid reveals the organism in these cases. Infection in infants is rapidly fatal; the child is infected *in utero* or during delivery, the mother showing no symptoms in many cases, although harbouring the organism. The most important single factor in the recognition of the disease may well be an awareness of the disease. The organism is easily isolated and recognized.

The method of transmission to man is not known. Wild animals and wild birds have natural infections, which are transmitted to domestic animals and from these, directly or indirectly, pass to man. The death rate in recognized infections in man was very high prior to the introduction of sulphonamides and antibiotics. Even with these it is about 30%. Cases have been described from many parts of the world, and the disease probably exists in Australia; but as there are no outstanding symptoms or signs, diagnosis depends largely on a recognition that there is such a disease.

INCIDENTAL SYSTOLIC HEART MURMURS.

Herbert Spitzbarth, in the *Deutsche medizinisches Wochenschrift*, June 14, 1957, has contributed an important article on what he calls *akzidentelle* systolic murmurs. These are heard chiefly over the pulmonary area, but occasionally in other precordial sites, and have no pathological significance whatsoever. Spitzbarth makes the rather surprising statement that most of the systolic murmurs heard in childhood and early adult life are of this type, which he distinguishes from so-called functional murmurs, in which there is some mitral valve incompetence or compression of the pulmonary artery. The differential diagnosis is not easy and demands all the resources of modern cardiography, particularly phonocardiograms taken in conjunction with electrocardiograms and the Röntgen profile. So common is this non-pathological sign that Spitzbarth agrees with Sahli that its absence calls for explanation more than does its presence. These incidental murmurs are heard in association with fever, hyperthyroidism, anæmia and effort. A laborious investigation has shown that the murmurs are loudest in the subjects concerned when the peripheral resistance is lowest and stroke volume highest. The cause is simply turbulence in the blood leaving the right ventricle or occasionally the left. Spitzbarth pleads for ampler recognition of the non-pathological character of these murmurs. He might have added that many healthy young men have been refused insurance or been allowed it only with loaded premiums because of such innocent signs.

[1] "Biological Aspects of the Transmission of Disease", edited by C. Horton-Smith; 1957. Edinburgh and London: Oliver and Boyd for the Institute of Biology. 8½" × 5½", pp. 194. Price: 36s. 9d.

[2] *M. Press*, 1957, 238: 206 (August 28).

[1] *Am. J. Clin. Path.*, 1957, 28: 18 (June).

Abstracts from Medical Literature.

PATHOLOGY.

The L.E. Test.

E. L. DUBOIS AND V. FREEMAN (*Blood*, July, 1957) have performed simultaneously a battery of different types of L.E. tests on peripheral blood drawn from one venepuncture in order to compare various techniques for doing the test. Their conclusions were that three types of test should be performed in order to secure the maximum number of positive results in systemic *lupus erythematosus*. The tests recommended are the sieved clot method, the Snapper-Nathan ring method and the Zinkham-Conley rotary glass bead technique. The details of these tests with improvements upon the originals are given. The largest number of positive results were obtained by the rotary method, and if only one test is to be done, this is their choice. They found that excessive amounts of heparin depressed the L.E. phenomenon. The mechanism of the test and the effect of treatment upon the test in their series are discussed.

Anæmia in Rheumatoid Arthritis.

E. J. FREIREICH, J. F. ROSS, T. B. BAYLES, C. P. EMERSON AND S. C. FINCH (*J. Clin. Investigation*, July, 1957) have used radioactive iron and red cell survival techniques to investigate the anæmia of rheumatoid arthritis. They conclude that there is a defect in the release of iron from the reticulo-endothelial system resulting in abnormally low plasma iron values. As a result of this, red cells are not produced in sufficient numbers, though iron utilization for erythrocyte production is normal and the mean corpuscular hæmoglobin values in these patients are only slightly lower than in normal subjects. Although red cell destruction and red cell production are both increased, red cells from a patient with rheumatoid arthritis survive for normal periods in normal subjects, indicating that the fault in rheumatoid arthritis lies in the red cell environment and not in the red cell itself. No reason for the increased hæmolysis was found.

Erythrocytes in Favism and Drug-Induced Hæmolytic Anæmia.

A. SZEINBERG, C. SHEBA, N. HIRSHORN AND E. BODONYI (*Blood*, July, 1957) have investigated acquired hæmolytic anæmia, particularly that due to ingestion of *Vicia faba* and various drugs. They found that in Israel, hæmolytic anæmia due to one of these causes occurs only in Sephardic Jews. This also applies to *thalassæmia major*. Ashkenazic Jews appear to be quite immune to these two groups of disorders. Various studies upon the erythrocytes of patients with past histories of acute hæmolysis due to favism, "sulpha" drugs and PAS showed no abnormalities of osmosis, mechanical fragility, electrophoretic mobility of hæmoglobin or its amino acid content. Glycolytic and catalase activities also were normal. The only constant abnormality was a deficiency of glutathione in the erythrocytes. Glutathione deficiency was also frequently found amongst the relatives of the patients. Although patients with *thalassæmia major* belonged to the same ethnic group, they all had an adequate glutathione content in their erythrocytes.

Osteolathyrism.

H. SELYE AND K. NIELSEN (*Arch. Path.*, September, 1957), in continuing the study of nitrile-induced bone disorders, have found that alizarin and kernechtrol, dyes with a particular affinity for osseous tissue, intensify the activity of aminoacetonitrile in the production of osteolathyrism. The disease is characterized by marked periosteal new-bone formation and disintegration of the bone canaliculi and of the junction cartilage plates, as well as slipping of the epiphyses. In the dosage used, neither of the dyes alone caused any demonstrable effect upon any organ, and the dosage of aminoacetonitrile was such that it alone caused little if any damage to the skeletal tissues of the rat.

Localization of Tuberculous Infection at Site of Injury.

J. W. S. BLACKLOCK AND J. R. B. WILLIAMS (*J. Path. & Bact.*, July, 1957) produced a tuberculous bacillæmia in rabbits and in guinea-pigs by inoculation into the left ventricle of suspensions of human tubercle bacilli (H.37Rv). Tubercle bacilli were found in the urine within two hours of infection. The infection was found to localize frequently at the site of injections of penicillin or peptone into muscles or joints, and around ligatures, but very rarely at the site of saline injections. Primary lung foci were produced in rabbits and in guinea-pigs by direct inoculation of tubercle bacilli into the lung, and by suitable timing of injections of penicillin into muscles the infection could be localized at the injection site in a small proportion of cases. Large doses of penicillin administered intravenously to tuberculous rabbits did not aggravate the infection. Injections of peptone or of preparations of penicillin into the muscles or into the peritoneum of normal animals produced a slight local mononuclear leucocyte reaction. When guinea-pigs were inoculated into the left ventricle with human tubercle bacilli, the subsequent intraperitoneal injection of penicillin or of peptone caused mononuclear leucocytes which had phagocytosed tubercle bacilli to appear in the peritoneal fluid within 24 hours. This mechanism is the likely explanation of the localization of the infection where there is damaged tissue or at the site of injection of foreign substances such as penicillin or peptone.

Bone Marrow Treatment of Radiation Injury in Mice.

C. C. CONGDON AND I. URSO (*Am. J. Path.*, July-August, 1957) have continued their investigations of survival rates amongst lethally irradiated mice receiving homologous bone marrow intravenously. They found that whereas untreated animals died within the first two weeks after exposure, animals treated with either homologous or isologous marrow showed only a decline in body weight for a few days followed by a parallel recovery. In the second two weeks the isologous animals gained weight and continued their recovery. The homologous mice, however, showed a secondary decline, during which many died. A few animals survived 107 to 316 days and, in general, resembled mice treated with isologous bone marrow. The histological picture in those mice which succumbed indicated a severe metabolic disturbance which the authors ascribed to an immune reaction. They suggest, too, that in view of the fact that some animals escaped the delayed homologous bone marrow reaction, it may be possible to circumvent the reaction under suitable conditions.

Influence of Muscle Section on Exostoses of Lathyric Rats.

C. J. HAMRE AND V. L. YEAGER (*Arch. Path.*, October, 1957) sectioned certain muscles of rats' hind limbs in order to determine the influence of muscle tension in the formation of exostoses in animals fed on a diet of sweet-pea seeds. Exostoses failed to appear at the insertions of the transected muscles, though they developed at the insertions of intact muscles. Their experiments showed that in addition to the sweet-pea factor, the presence of muscular tension is required for the development of exostoses.

Alterations of Elastic Fibres in Lathyric Rats.

D. G. WALKER (*Arch. Path.*, October, 1957) has found evidence of widespread arterial involvement in young rats treated with the lathyrogenic factor of sweet-pea, beta-aminoproprionitrile. The extent to which the arterial system becomes involved depends upon the age of the animal. To produce elastic fibre alterations elsewhere than in the aorta, the toxin must be administered during the first few weeks after birth, when elastogenesis normally is particularly active. Rats over seven weeks of age are not subject to aortic aneurysm formation. This indicates that the disturbance is due to inhibition of elastic formation rather than destruction of elastic fibres. There was, however, an increase in fibrous tissue and also in the argyrophilic precursor of elastic. In the walls of all major arteries there was also a deficiency of mucopolysaccharides. Elastin formation was inhibited in a variety of other tissues, including the matrix of the external auditory canal cartilage.

Scleroderma of the Gastro-Intestinal Tract.

M. B. GOLDGRABER AND J. B. KIRSNER (*Arch. Path.*, September, 1957) have reviewed the subject of scleroderma of the gastro-intestinal tract. The œsophagus is the most commonly involved, and the lesions may be thickening or thinning of the wall and inflammation with ulceration and stenosis of the distal portion. Most authors regard the ulceration and stricture as secondary to regurgitation of acid gastric juice. Histological diagnosis by the examination of biopsy material is not usually possible. Changes in the stomach are less common. They consist in submucosal fibrosis with or without inflammation and ulceration. In the small intestine, the most frequently described changes are muscular atrophy, fibrosis, lymphocytic infiltration and saccular dilatation. In three of the reported cases there was duodenal ulcer. The colon may be involved in scleroderma and the recorded manifestations are dilatations or sacculations, atrophy of the

muscularis, submucosal sclerosis, lymphocytic infiltration and ulceration. In several instances there was similarity with the picture of ulcerative colitis, but scleroderma differed in affecting shorter segments of bowel.

Accessory Spleens.

B. HALPERT AND F. GYORKEY (*Arch. Path.*, September, 1957), in 2250 consecutive autopsies at the Veterans Administration Hospital, found accessory spleens in 222 instances (almost 10%). One hundred and ninety-one had one accessory spleen, 25 had two, and six had three or more accessory spleens. The 191 solitary accessory spleens were located as follows: 147 at the hilus, 31 in the tail of the pancreas, six near the hilus, six along the splenic artery, and one in the wall of the jejunum. Among the 25 subjects with two accessory spleens, both were located at the hilus in 19; in three, one accessory spleen was in the tail of the pancreas and the other at the hilus; in two, one was at the hilus and the other nearby; in one subject both accessory spleens were in the tail of the pancreas. Of the six subjects with three or more accessory spleens, they were located about the hilus or along the splenic artery in five, and in one there was an accessory spleen in the tail of the pancreas and the remainder were nearby. Thus an accessory spleen in the tail of the pancreas was observed in 36 of 222 subjects with accessory spleens—an incidence slightly over 16%.

THERAPEUTICS.

Enterobiasis.

T. S. BIMBALO *et alii* (*J.A.M.A.*, August 10, 1957) describe the treatment of enterobiasis with one oral dose of promethazine hydrochloride ("Phenergan"). One hundred and seven children infected by the pin worm (*Enterobius vermicularis*) were treated with 125 milligrammes of "Phenergan" in a single dose at bedtime. Ten days later only five of the 107 children gave consistently negative results over a seven-day period. A second series of 24 patients were treated with 125 milligramme doses of pyrathiazine. The percentage of successes was 12·5. Larger doses of the drugs were not without risks of side effects. These results suggest that other more effective oxyuricides of established value should be used instead of these substances.

Acne Vulgaris.

D. TORRE AND M. M. KLUMP (*J.A.M.A.*, July 27, 1957) describe cyclic œstrogenic hormone therapy of *acne vulgaris*. Seventy-one female patients between the ages of 13 and 30 years were treated by oral administration of œstrogens. There is some evidence that androgens may help to cause acne—for instance acne is said to be more frequent in young men—and that œstrogens may relieve acne, e.g. in pregnancy and in the middle of the menstrual cycle, when œstrogen levels are highest. The patients treated were investigated by a vaginal smear technique. Then a conjugated œstrogenic preparation was given orally for 14 days before the expected date of the menses. One and a quarter milligrammes per day of this preparation were given, and with each succeeding menstrual cycle the dose was raised by 1·25 milligrammes per day until a good clinical result or the vaginal smear showed adequate physiological response. The dose was then decreased by 1·25 milligrammes in succeeding cycles, the average dose being 3·9 milligrammes daily. In general, the results of treatment were good in 57%, satisfactory in 31%, and poor in 12%. The average length of treatment was six and a half months.

Intramuscular Use of Iron.

A. B. HAGEDORN (*J.A.M.A.*, August 10, 1957) discusses the intramuscular use of iron in the treatment of iron-deficiency anæmia. "Imferon" (Benger), a stable non-irritant solution of iron-dextran complex, was injected into the gluteal muscles twice daily in doses of 2·0 cubic centimetres, said to contain 100 milligrammes of iron, in 10 patients with hypochromic anæmia due to gastro-intestinal or general bleeding. The iron was proved to be absorbed, the anæmia improved rapidly, and there were no adverse effects such as pain or systemic reactions. In the 10 patients treated the response was satisfactory, preferable to intravenous iron therapy, but not generally preferable to oral administration of iron in this type of anæmia. The conclusion was that oral administration of iron was most reliable in iron-deficiency anæmia, but that iron given intramuscularly could be used as an alternative or as a supplement.

NEUROLOGY AND PSYCHIATRY.

Treatment of Chronic Alcoholism.

R. S. WALLERSTEIN (*Am. J. Psychiat.*, September, 1956) for over two and a half years studied 178 alcoholic patients divided into four more or less equal groups, each group being subjected to a different mode of therapy, as follows: (i) "Antabuse"; (ii) conditioned reflex; (iii) hypnotherapy; (iv) group therapy. A period of approximately three months was set as the limit of time for close supervision. The "Antabuse" group complied most successfully with the therapy, the patients' contribution being mainly restricted to the taking of pills. For some patients, certainty of enforced sobriety can be dangerous. In this group are the borderline schizophrenics and those warding off deep depressive reactions. Conditioned reflex therapy was most successful when active cooperation was obtained by presenting the treatment as a partial barrier requiring constant support by the patient. The more it was represented as an absolute barrier, the more was the tendency to test its effectiveness. The third group was treated by hypnotherapy. The alcoholic was quite hypnotizable, and it was found that the more hypnotizable he was, the more likely was he to complete treatment. The passive dependent personality did best with this mode of treatment. In the more aggressive type there was greater difficulty in being hypnotized, and a greater number of failures resulted. The fourth group was given group therapy. The author concludes that "Antabuse" is more helpful to more patients than any other treatment. The interpersonal relationship between the therapist and patient is important in any form of treatment. The strongly aggressive patient poses difficulties in any group.

Schizophrenics in the Navy.

C. W. WAHL (*Am. J. Psychiat.*, September, 1956) has determined the frequency of various antecedent familial experiences in the histories of 568 schizophrenics from the United States Navy, and has compared this with incidence data from the general population, from a group of 100,000 naval recruits and, when applicable, from a previously studied group of 392 schizophrenics from the Elgin State Hospital. The factors investigated were: (i) marked rejection and/or over-protection by one or both parents; (ii) bereavement by death or separation of one or both parents prior to the fifteenth year; (iii) placement in the sibling hierarchy; (iv) family size; (v) religious affiliation. It was found that among this group of schizophrenic navy personnel, 50·3% came from homes in which there was severe rejection and/or over-protection by one or both parents. Severe rejection by one or both parents was by far the most frequently manifested attitude, 34% of subjects having been so rejected, while only 9% were over-protected by one or both parents. Of the entire group, 41% had lost a father or mother by death, divorce or separation before the age of 15 years, as compared with 11·4% among the generality of navy men. Approximately four times as many of the schizophrenic group had been orphaned prior to their fifteenth year as in the general population. As to sibling position, it was found that youngest children slightly, but definitely, predominated. The average family size of members of the schizophrenic sample was 4·4 children per family. This compares with a national average of 2·2 per family. It is held that large family membership may produce specific stress situations.

The Ataraxic Drugs.

H. A. BOWES (*Am. J. Psychiat.*, December, 1956) states that the ataraxic drugs are so called from their ability to produce ataraxy—freedom from confusion and anxiety. The present observations were made in a veterans' psychiatric unit of 550 beds with a high rate of admission and discharge. "Frenquel" appears to have an anti-hallucinatory effect on a small number of schizophrenics, but is of no value in the treatment of acute psychoses. Reserpine is reserved for the tranquillizing of arteriosclerotics and as an adjunct to psychotherapy in some withdrawn schizophrenics. "Pacatal" and chlorpromazine are virtually interchangeable and synergistic in action. Both are helpful in cases of psychomotor excitation, aggression, destructiveness, restlessness and tension, in both acute and chronic psychoses. These drugs have significantly reduced the need for electroshock therapy and lobotomy without replacing these valuable methods of treatment. The author states that in his experience insulin coma has been replaced by group psychotherapy with the use of ataraxics as adjuncts. Ataraxics merely modify symptoms, but when they are used in conjunction with psychotherapy, some fundamental therapeutic improvement may be expected.

XUM

Brush Up Your Medicine.

VAGINAL DISCHARGES: LEUCORRHŒA OF VAGINAL ORIGIN.

VAGINAL DISCHARGE, like the common cold, is a frequent and troublesome complaint which occurs at all ages, and which is variable in its significance and response to treatment. The term leucorrhœa will signify in this paper an abnormal, non-hæmorrhagic discharge not caused by neoplasm or other serious organic disease.

The genital tract is normally moistened by glandular secretions from particular organs such as the vulva, *cervix uteri*, uterus and Fallopian tubes. The vagina, however, is devoid of glands, but is bathed by secretions from above, particularly the cervix, and is also moistened by transudation through its epithelial lining. Any discharge which makes the patient constantly aware of being moist, or which is enough to stain the underclothes, is abnormal.

Normal vaginal secretion is scanty, and is white or grey in colour and of granular consistency. It consists of desquamated epithelial cells containing glycogen, cervical mucus and the lactobacillus of Döderlein. Non-pathogenic organisms including staphylococci, streptococci, diphtheroids *etc.* are present in 50% of normal vaginal secretions. The reaction of the normal secretion is acid, with a pH of 4·5 to 5·7. There is marked personal variation in the normal discharge, apart from physiological changes associated with age and the influence of the sex hormones. Doubt exists as to whether the vaginal epithelium is developed wholly or in part from the Müllerian ducts or from the uro-genital sinus.[1] There is also a difference of opinion concerning cyclic changes in the human vagina. There is no doubt, however, that the vaginal epithelium is responsive to the stimulus of the ovarian hormones, particularly œstrogen.

An understanding of the physiology of the vagina is essential for the assessment and treatment of vaginal discharges. This includes consideration of the nature and changes of the vaginal epithelium in response to the sex hormones, the glycogen content of the epithelial cells, the pH of the vaginal secretion and the presence or absence of Döderlein's bacilius. The vaginal epithelium is stimulated and built up under the influence of œstrogen and is rich in glycogen. Glycogen is broken down by non-bacterial enzymes into sugars, which in turn are converted into lactic acid by the action of Döderlein's bacillus. A normal acid pH is thus maintained. Interference with the growth and glycogen content of the epithelium and interference with the normal bacterial flora and pH of the vaginal secretion are disposing factors towards vaginal disease and abnormal discharge.

The vaginal epithelium of the newborn baby is thick and rich in glycogen under the influence of maternal œstrogen. Döderlein's bacillus are found in the vagina within a day or two of birth, and the secretion is acid in reaction. In childhood, to the age of puberty, the œstrogen stimulus is lacking; the epithelium degenerates, glycogen disappears, Döderlein's bacilli are absent and the secretion is alkaline. During reproductive life, hormonal influence is active and normal biological conditions are maintained. After the menopause ovarian activity wanes, the epithelium again becomes thin, glycogen disappears, the normal flora may be absent and the secretion may be neutral or even alkaline (pH 6 to 7). The administration of œstrogens to patients in the two age groups with deficient œstrogen stimulus promptly changes the epithelium and secretion to that of the normal vagina.

EXAMINATION OF THE PATIENT.

The essential points in routine examination and investigation of the patient may be summarized as follows:

The history should cover the patient's age, marital state, parity and past history. Inquiry should be made as to the onset and duration of the discharge and its character (amount, colour, odour, consistency), and also as to the relation of the discharge to menstruation, ovulation and coitus.

The patient's weight should be recorded and her urine examined. A general physical examination should cover all systems. A full pelvic examination should be made, as follows: (i) vulval inspection (Skene's ducts and glands, Bartholin's ducts and glands, discharge); (ii) vaginal and bimanual examination (no lubricant); (iii) speculum examination (good light). The pH of the discharge should be tested with litmus paper, and a drop should be examined immediately (warm slide, warm saline, coverslip, high power of the microscope). A rectal examination should be carried out, especially in children and virgins. Special investigations are usually necessary. Specimens of the discharge should be collected for bacteriological examination. Urethral and cervical smears should be prepared and cultures attempted, and a saline suspension of the discharge should be made. A smear should be prepared for staining by Gram stain or methylene blue. Blood tests include the Wassermann test and the Neisserian complement deviation test.

A careful history often gives an immediate lead to the diagnosis. Routine examination appraises the patient's general health, the local condition of the pelvic organs, and the character and source of the discharge. Virginal patients can usually be examined with suitable small specula. Examination under anæsthesia is rarely necessary except when a foreign body is suspected in children. A nasal speculum, urethroscope or Kelly's cystoscope may be helpful in such cases. The left lateral or knee-chest position may be used with advantage in difficult cases. It is usually possible to make a diagnosis in most cases of vaginal leucorrhœa by routine pelvic examination and immediate hanging drop examination. This procedure is within the scope of any general practitioner or gynæcologist.

LEUCORRHŒA OF VAGINAL ORIGIN.

The following is a brief classification of leucorrhœa of vaginal origin:

Physiological leucorrhœa: normal vaginal biological picture.

- Simple hypersecretion.
- Leucorrhœa occurring before and after menstruation (uterine origin).
- Leucorrhœa occurring at ovulation (cervical origin).
- Leucorrhœa due to sexual excitement (Bartholin's glands and cervical origin).
- Constitutional leucorrhœa, due to debility, fatigue, malnutrition, anæmia.

Pathological leucorrhœa: changed vaginal biological picture.

- (*a*) Leucorrhœa due to infection—gonococcal, trichomonal, monilial.
- (*b*) Leucorrhœa due to infection and endocrine imbalance—bacterial vulvo-vaginitis of childhood, senile vaginitis.

Leucorrhœa due to miscellaneous causes.

- Foreign bodies.
- Chemical irritants.

Physiological Leucorrhœa.

The borderline between normal and excess vaginal discharge is not easy to determine. The slightest discharge may assume abnormal significance to the ultra-fastidious patient. A guilt complex or the fear of uncleanliness, venereal disease or cancer may cause a psychosis without the existence of organic disease. Other patients neglect gross discharge until it becomes blood-stained or offensive and signals the presence of genital cancer. Physiological leucorrhœa may be associated with chronic ill health and may date from the menarche and follow a cyclic pattern. It usually occurs in women who have not been pregnant and have no past history or evidence of genital infection. The discharge is normal in appearance and has a normal pH and vaginal flora. Immediate hanging drop examination shows desquamated epithelial cells and no pus cells. The clear, watery discharge before and after menstruation and the clear mucoid discharge in some women at the time of ovulation are well recognized. During pregnancy there is a general increase of the normal white discharge, which has the appearance of thin condensed milk. Sexual excitement is usually accompanied by the outpouring of a clear mucoid discharge from Bartholin's glands and the cervical glands. Constitutional diseases, fatigue, malnutrition and nervous disorders may in some way alter the physiology and result in leucorrhœa. The diagnosis of a physiological discharge is readily made from the history, routine examination and immediate drop examination. Treatment is directed towards reassuring the patient by simple explanation, attending to any constitutional disease and endeavouring to correct any emotional disturbance. Local treatment is avoided on account of the risk of interference with normal conditions in the vagina.

Pathological Leucorrhœa.

Gonococcal Infection.

There has been a marked decrease in the incidence of gonococcal discharge during the past ten years. The well-developed squamous epithelium of the vagina during the reproductive years is resistant to gonococci and other germs. The poorly developed epithelium present before puberty and after the menopause is prone to infection. Gonorrhœa in the female adult favours sites other than the vagina, notably the urethra, Skene's glands and ducts, Bartholin's glands and ducts, the cervical glands and the uterine tubes. In acute gonococcal infection there is usually a history of recent exposure, followed quickly by acute urinary symptoms and the occurrence of a yellow, purulent discharge. The presence of acute urethritis, redness and palpable thickening of Skene's and Bartholin's ducts and acute cervicitis is of great diagnostic significance. Chronic gonococcal infection may be difficult to diagnose. Evidence of chronic infection is sought at the sites previously mentioned. The diagnosis of any suspected gonococcal infection is not established until it has been confirmed by bacteriological examination of smears and cultures. The treatment of Neisserian infection is now on a specific basis with penicillin, which can be given by injection in doses of about 600,000 units daily for five days. Chronic foci of infection in the cervix, Skene's glands and ducts and Bartholin's glands and ducts may require excision, cauterization or diathermy as indicated.

Trichomonas Vaginalis Vaginitis.

Trichomonas vaginalis vaginitis is the most common cause of leucorrhœa after puberty, and is no respecter of persons, race, marital state or social state. It would be difficult to assess the physical and mental discomfort caused by this disease. The protozoon was first described by Donné in 1836, but we still lack accurate knowledge of the nature of trichomonal infection and of its prevention and specific treatment. *T. vaginalis* is a motile, pear-shaped protozoon intermediate in size between pus cells and epithelial squames as seen in wet-drop examination of discharge. Only half the women who harbour trichomonads have vaginitis and discharge. Some added factor has been suggested to explain the behaviour of the organism as a pathogen. Secondary infection, symbiosis with streptococci, altered vaginal physiology and psychogenic factors have been considered predisposing causes. It has been shown that the introduction of pure cultures of *T. vaginalis* into a healthy vagina causes the typical vaginitis. Infection is thought to be transmitted by fingers, towels, toilet seats, baths and vaginal appliances such as douche nozzles and pessaries. Primary infection and subsequent reinfections would appear to be frequently transmitted by sexual intercourse. The menstrual flow favours the growth of trichomonads, and symptoms are marked and relapses common after menstruation. Persistent reinfection is relatively common, and may arise from endogenous foci such as the cervical glands, Skene's glands and Bartholin's glands. Exogenous foci may include infected urethral and prostatic discharge of the male partner in addition to the possible sources enumerated.

The symptoms and signs of trichomonal vaginitis are as follows. There is a copious, thin discharge, of cream, yellow or green colour, characterized by an acrid odour and the presence of frothy bubbles. The vulva may be inflamed, and the patient may complain of pruritus and burning. Associated urinary symptoms and dyspareunia may or may not be present. The vaginal vault and cervix are usually studded with small, red, punctate spots giving the characteristic "strawberry" vagina. The cervical canal is not infected, nor can discharge be expressed from the posterior part of the urethra as in Neisserian infection. The diagnosis is confirmed by the simple procedure of immediate hanging drop examination of the discharge under the microscope. The presence or absence of the protozoon and the ratio of pus cells to epithelial cells are noted.

Probably no gynæcological disease has received such a wide variety of treatment as trichomonas vaginitis, which indicates that a specific cure is unknown. Broadly speaking, 75% of patients can be cured by routine treatment, the condition can be controlled in 20%, and in 5% it is resistant to treatment. Many women seem to develop this disease while away from their own home environment, and recurrent infection appears to be more common in the presence of physical illness or emotional upset. Women who are away from home would be well advised to take showers rather than baths, and to take precautions against infection from toilet seats. The equipment with gap-seats of all public toilets for women is desirable in prophylaxis against trichomonal and monilial infection. It is desirable to continue the treatment of trichomonal vaginitis through menstruation and over the next four days for at least three successive menstrual periods. The nature of the complaint and possible difficulties in treatment should be explained to the patient in order to secure her cooperation.

A combined method of treatment such as the following has been found generally satisfactory. The vulva and vagina are thoroughly cleansed, and the vagina is insufflated with "Compound Picratol Powder". The patient is instructed in the use of silver picrate suppositories ("Picratol"), and insufflation is repeated after one week. The suppositories are continued for another week, and the patient's condition is assessed a few days later. Treatment is continued over the three next menstrual periods and the succeeding four days by inserting acetarsol vaginal compound ("S.V.C.") tablets into the vagina night and morning. If the response to silver picrate treatment has not been prompt, a change is made to another type of treatment such as "Floraquin" tablets, moistened and inserted into the vagina by the patient night and morning. In difficult cases with relapses, an investigation of possible endogenous or exogenous foci of infection is required. Examination of the husband may indicate the temporary avoidance of coitus or the use of a condom. Consideration of the patient's general health and the treatment of any chronic disease or nutritional disturbance are indicated. The conception of trichomonal vaginitis as a psychosomatic disorder merits consideration. The trichomonad is considered the specific infective agent, but is thought to be pathogenic only when the vagina is influenced by the effects of emotional disturbances. Treatment along these lines is by a minimum of local measures and simple methods of psychotherapy.

Monilial Vaginitis.

Monilial infection ranks next to trichomonas infection as a cause of vaginitis. *Candida albicans* is the only one of many species of yeast fungi which is pathogenic in man. It is found infecting the mouths of babies, the skin, the nail-beds and the respiratory tract. The fungus thrives in an acid medium (pH less than 4·5), and is commonly found in pregnancy and in patients with glycosuria. An increased incidence of mycotic disease of the vagina has been noted in patients under treatment with certain antibiotic drugs. These drugs alter the normal vaginal flora and conditions, and favour the growth of monilia. *C. albicans* is identified by its numerous tiny budding spores attached to pale mycelial filaments. It may be seen in wet-drop examination, but diagnosis is easier from a smear stained with Gram's stain or methylene blue. In doubtful cases, cultural investigation of the fungus is the most accurate method of diagnosis. *C. albicans* may be present in the vagina without causing discharge or other symptoms. The factor which determines its pathogenic activity is not known. Infection is thought to be transmitted by fingers, towels, baths, toilet seats, infected vaginal appliances and coitus.

The chief symptoms of monilial vaginitis are vulval irritation and a scanty white discharge, usually of sudden onset. The vulval irritation and burning are usually out of proportion to the amount of discharge noticed by the patient. Dysuria and dyspareunia are variable symptoms. The vulval skin may appear normal, or it may be the site of excoriation or maceration. The discharge is usually most evident in the upper half of the vagina as white, flaky, adherent concretions, which have been aptly likened to milk curds or damp bread, on a dark-red vaginal wall. The removal of adherent discharge may cause small areas of bleeding. The discharge may not always present these typical features, but *C. albicans* infection should be considered in any resistant case of *pruritus vulvæ*. The endocervical canal is not infected, nor does the fungus lodge in the urethra or in Bartholin's ducts. Microscopic confirmation of the diagnosis is often unnecessary in the presence of typical clinical findings.

The treatment of monilial vaginitis is on a more satisfactory basis than that of trichomonas vaginitis. However, mycotic infection in late pregnancy may be difficult to control. The urine should be tested, and any condition of glycosuria requires investigation and treatment. The so-called diabetic vulvo-vaginitis is usually due to associated moniliasis and does not settle until the metabolic disturbance is controlled. Hesseltine has suggested that every diabetic with vulvitis should be examined for mycotic vaginitis, and every patient with mycotic infection should be examined for *diabetes mellitus*. A routine treatment comprising initial vaginal paintings with gentian violet followed by vaginal insertion of propionate jelly has been found satisfactory. The vagina is thoroughly cleansed and painted with a

saturated aqueous solution of gentian violet (2%) three times during the first week. The patient is then instructed in self-medication with "Propion Gel". Five cubic centimetres of the jelly are introduced into the vagina through an applicator night and morning for two weeks. Treatment with gentian violet pessaries is not favoured on account of the frequency of chemical vaginitis. Mycotic dermatitis of the vulva secondary to monilial vaginitis responds to treatment of the vaginal infection and the local application of hydrocortisone ointment (1% to 2%). The husbands of patients with recurrent monilial vaginitis should be examined, and any vaginal appliances such as douche nozzles or diaphragms should be carefully sterilized. Prevention of infection from toilet seats and baths applies as in the case of trichomonal vaginitis.

Vulvo-Vaginitis of Childhood.

Vulvo-vaginitis is prone to occur in children before the age of puberty, when the squamous epithelium of the vagina is under-developed and offers poor resistance to germ infection. Probably less than half these cases are gonococcal in origin, the remainder being due to mixed non-specific germ infection. The vulva is affected chiefly in the region of the *labia minora*. The urethra, Skene's ducts and glands and Bartholin's ducts and glands are not usually involved. The vaginal wall may be inflamed throughout its entire length and thickness. Infection may be carried by fingers, clothes, towels, toilet seats and baths, and the disease may spread through an institution like an epidemic. The symptoms may be acute, but more commonly a chronic discharge which stains the underclothes is noticed by the parents. The following conditions are considered in the differential diagnosis: gonococcal vulvo-vaginitis, non-specific infection occurring in neglected children, non-specific infection following irritation due to worms or to a foreign body in the vagina. The diagnosis of gonococcal vulvo-vaginitis is established by identification of gonococci by examination of smears and by culture.

The treatment of gonococcal vulvo-vaginitis is with the specific antibiotic, penicillin, in doses of about 300,000 units per day for five days. In non-specific infections the organisms are tested for antibiotic sensitivity and the suitable antibiotic is given. Local treatment is limited to frequent cleansng of the parts. In resistant cases of specific or non-specific vaginitis, any predisposing cause such as worms or foreign bodies is to be excluded, and œstrogens are given by mouth, as well as the appropriate antibiotic. A suitable dose of œstrogen is one "Dienœstrol" tablet (0·3 milligramme) thrice daily for ten days.

Senile Vaginitis.

Senile vaginitis is primarily an atrophic change of the vaginal mucosa with altered biological conditions in the vagina and superadded secondary infection. The condition is found in post-menopausal women, and is more common when the menopause has been induced by operation or irradiation. The vaginal discharge is neutral or alkaline (pH 6 to 7), glycogen is absent from the epithelial cells, and Döderlein's bacilli may be absent. Small denuded and ulcerated areas occur in the mucosa of the upper third of the vagina, resulting in a red, mottled appearance. A watery or cream discharge makes its appearance, and may become blood-stained as infection develops and granulations may form in the vault. The sequel of this is the development of either adhesions or scar tissue, which may cause vaginal contraction and atresia.

Symptoms may be negligible, or the patient may complain of leucorrhœa, blood-stained discharge, *pruritus vulvæ*, burning pain in the vagina or dyspareunia. Contact bleeding and blood-stained discharge in such patients usually demand full gynæcological examination, including a biopsy of the cervix and diagnostic curettage to exclude uterine cancer.

The differential diagnosis of senile vaginitis includes carcinoma of the cervix, carcinoma of the body of the uterus, pyometra due to various causes and trichomonal vaginitis. Immediate hanging drop examination reveals a predominance of pus cells over epithelial cells, and a change from the usual cornified cells to round basal epithelial cells.

Vault adhesions and contracture of the vagina without discharge or other subjective symptoms do not require treatment. Patients requiring treatment respond to the local insertion of œstrogen pessaries and small controlled oral doses of œstrogens.

Miscellaneous Causes of Leucorrhœa.

Mention has been made of foreign body in the vagina as a possible cause of vaginal discharge in children. Any discharge in a child which is not a gonococcal condition and which does not respond promptly to treatment should be considered due to foreign body until proved otherwise. It may be possible to feel the foreign body by rectal examination, but examination under anæsthesia is usually necessary. Foreign bodies causing vaginal discharge in adults are discovered from time to time in normal women as well as in the mentally deranged and perverted types. Forgotten internal pads, contraceptive appliances and pessaries are the most commonly found articles. The discharge disappears soon after removal of the cause and treatment by a few cleansing douches.

Chemical irritation due to over-zealous local treatment, strong drugs or sensitivity on the part of the patient is not uncommon. Gentian violet pessaries and locally applied antibiotics may occasionally cause vaginitis with discharge.

Summary.

An understanding of the physiology of the vagina is essential for the assessment and treatment of vaginal discharges. Interference with the growth and glycogen content of the vaginal epithelium and change in the normal bacterial flora and in the pH of the vaginal secretion are predisposing factors towards vaginal disease and abnormal discharge.

A careful history, thorough physical examination and immediate hanging drop examination of the discharge permit prompt diagnosis of most cases of vaginal leucorrhœa.

Trichomonas vaginitis is the most common cause of leucorrhœa and is the most difficult to treat on account of lack of knowledge concerning its nature, mode of transmission and tendency to reinfection.

It would appear that both trichomonal and monilial vaginitis may be acquired from contaminated toilet seats and baths. This suggests a possible means of prevention of infection. In difficult relapsing cases, foci of infection are sought and the husband should be examined as a possible carrier.

Conditions such as chronic illness, fatigue, malnutrition and emotional disturbance appear to dispose patients towards persistent and recurrent discharge.

Vulvo-vaginitis of childhood and senile vaginitis are considered as infections of under-developed and atrophic vaginal epithelium associated with altered vaginal physiology and poor resistance to infection.

The principles of treatment of any type of leucorrhœa aim at eliminating the causative organism, restoring normal vaginal physiology and preventing reinfection in the simplest and most acceptable way to the patient.

A. A. MOON.

Sydney.

Obituary.

MERVYN ARCHDALL.

MERVYN ARCHDALL, second Editor of THE MEDICAL JOURNAL OF AUSTRALIA, died on September 6, 1957, six days after his official retirement from the editorial chair. He was aged 73 years.

Born on August 31, 1884, at St. Mary's Parsonage, Balmain, Mervyn Archdall was the son of an Irish father and a German mother. His father, the Reverend (later Canon) Mervyn Archdall (the name Mervyn is a family tradition), was born in the south of Ireland, and was educated at Durham Grammar School and Corpus Christi College, Cambridge. He was a Hebrew, Greek and Latin scholar, and also won a prize for English literature. When he came to Australia he brought as his young bride Martha Caroline Christine, fifth daughter of Pastor William Adolphe Korow, who served at a little place called Teschendorf in Pomerania. This remarkable woman, whom Mervyn always referred to with great affection as his "little mother", had become a teacher on the death of her father, and in later years used her teaching ability for the benefit, among others, of her three children. In 1888 the Reverend Mervyn Archdall, in fulfilment of a promise, took his wife back to see her mother, and the two small children, Mervyn and his younger brother Henry, went too. According to his own account, during the four months that he was in Germany, Mervyn learned to jabber German and forgot his English; he always regretted that his German was allowed to lapse when he returned to Australia, and he later had to re-learn it the hard way. His

main memory of the trip seems to have been the four-candle birthday cake at the celebration of his fourth birthday in Stetten.

On the family's return to Australia, the question of schooling for Mervyn arose. He was first sent to the public school at Nicholson Street, East Balmain, which for a number of good reasons he hated. Then for a time his mother undertook his instruction with that of five other boys. A short term at St. Philip's Grammar School, Church Hill, ended when his father started a parochial day school under the control of Miss Clara Jane Squire. Miss Squire was a splendid teacher, who drummed English grammar into her pupils, and her basic teaching stood Mervyn in good stead long years afterwards in his editorial work. The next step was Sydney Grammar School, which made a lasting impression. Mervyn used to speak in especially high terms of the headmaster, A. B. Weigall, who with the mathematics master, A. H. S. Lucas, gave him special tuition to enable him to matriculate. Then, as always, Mervyn found mathematics a burden, but he succeeded in matriculating.

Financial difficulties stood in the way of the medical course that Mervyn had hoped for, and his father wished both his sons to take orders. However, at the last moment, a letter arrived from Mervyn's maternal grandmother, offering to pay his fees to study medicine, and he entered the University of Sydney. He studied hard, and to his surprise, in his first year topped the list in zoology, winning Professor Haswell's Prize, and shared the Renwick Scholarship with E. A. Brearley. In his subsequent years the teachers who impressed him most were J. T. Wilson, the Professor of Anatomy, and Robert Scot Skirving at the Prince Alfred Hospital (it was not then Royal). He finished the course near the top of the year, graduating M.B. in December, 1907.

As a resident medical officer at Sydney Hospital, Archdall came under the influence of such men as Jamieson, Maitland, Corlette and Fiaschi, of whom he often used to speak. More important still, he there met Head Nurse Mary Elizabeth Thompson, who later became his wife. On leaving the hospital, he spent a month as a *locum tenens* in order, as he said, to buy an engagement ring. Then followed a useful and enjoyable trip to Japan as a ship's surgeon, and a period in general practice at Winton in Central Queensland and at Charters Towers in North Queensland. He next bought a general practice in his old home suburb of Balmain, and found an opportunity to develop his surgical leanings as a member of the staff of the Balmain and District Hospital, and also during two periods as an honorary relieving assistant surgeon at Sydney Hospital.

This experience stood him in good stead when in January, 1917, he joined the A.I.F. and went to France. He was classed as a surgeon at the Second Australian General Hospital, where he worked under Upjohn, Hurley and Maclure. Subsequently he was given charge of a surgical team, which worked up and down the line and provided him with a great variety of surgical experience. He derived great enjoyment in his leisure time from helping with concert parties, and especially in training a small group of members of the hospital unit to sing part songs. He also sang the tenor part in Mendelssohn's *Lobgesang* with the musical society in Wimereux.

On his return to Australia, Archdall had to decide between taking up surgery or returning to general practice, and on advice chose the latter. After a few months at Stanmore, he went to Coonamble, but found that he was not cut out to be a country general practitioner. He often described the broiling summer afternoon in 1922 when he opened his copy of THE MEDICAL JOURNAL OF AUSTRALIA and saw an advertisement for an Assistant Editor. He immediately sat down and wrote to the Editor. He was not appointed, but left Coonamble just the same and went to Tasmania for a spell. As it happened, the successful applicant for the Assistant Editor's position decided after two weeks that it was not for him, and a telegram was sent offering the position to Mervyn Archdall. He was away touring, and was inaccessible, so his wife took the responsibility of accepting on his behalf, an action which he fully approved, and one for which the medical profession in Australia must be forever in Mrs. Archdall's debt. The position was at first officially half-time, but it soon proved to be more than that, and after twelve or eighteen months it was made officially full-time.

Archdall worked under the editorial guidance of H. W. Armit for eight years, and in 1930 succeeded him as Editor. The story of his editorial career and of the great contribution which he made to medicine in this country was told in a leading article on his retirement on August 31 of this year. It is unnecessary to go over this ground in detail again, especially as the tributes of others published in this issue relate mainly to that period. It is sufficient to say that he raised the Journal to a standard that ensured world-wide respect, and served the whole profession in whatever ways lay in his power. The Federal Council of the British Medical Association in Australia awarded him its Gold Medal less than a week before his death, and it may be said without reservation that this honour, which he prized above all else, was a recognition of unstinted and effective service, not only to the British Medical Association, but to medicine and its practitioners throughout Australia and to the whole community.

Mervyn Archdall's interest, knowledge and sympathies were wide and deep. His early days in Balmain had given him an insight into the lives of the less privileged members of the community, and he maintained a strong interest in social questions, always being ready to champion the underdog. At the same time he loved life and the good things of life. Most of all he loved medicine, both as a science and as an art concerned with serving ordinary men and women in their everyday need. His influence for good was probably far wider than he ever knew or could know.

SIR HENRY NEWLAND, Chairman of the Board of Directors of the Australasian Medical Publishing Company Limited, writes: During my unusually long term of office as President of the Federal Council I had a lengthy association with Dr. Archdall. He had a standing invitation from the Council to attend its sessions. He valued this privilege, and unless prevented by ill health was most punctilious in his attendance. He took ample notes of the debates and reinforced them, when necessary, in consultation with Dr. John Hunter, the Secretary, in whom he found a loyal ally. Consequently the proceedings of the Council were always well reported in THE MEDICAL JOURNAL OF AUSTRALIA. Archdall was trusted by the Council to refrain from publishing anything of a confidential nature, and that trust he never betrayed. The Federal Council is the governing authority of the Australasian Medical Congress, and it had become usual to entrust Archdall with the publication of the transactions in THE MEDICAL JOURNAL OF AUSTRALIA. This exacting task has been a boon to those who prefer to read the transactions in their MEDICAL JOURNAL rather than in a separate official publication.

But it is as chairman of the Board of Directors of the Australasian Medical Publishing Company Limited that I have come into much closer relation with the late Editor and been able to verify his great gifts. It has been a recurring and happy experience, too, to have passed the time of day with him in that semi-pontifical sanctum at "The Printing House" where Archdall exercised his high and wise authority. We used to meet, too, at his charming home "Grantchester" and there enjoy his hospitality, good company, good talk, good fare and good fun. Archdall the Editor was in no sense egotistical; he was really a modest man, but like many other great editors he had come to have a nice appreciation of the merits and standing of his own paper and of the dignity of its Editor. And rightly so. He lived and, at the end, died for that which he had spent himself to serve so well, THE MEDICAL JOURNAL OF AUSTRALIA.

DR. W. L. CALOV writes: In 1917, as fourth year students at Sydney Hospital, a group of us met in the outdoor department a young, handsome and efficient surgeon, who attracted us by his kindness and courtesy and his ability to teach. This was Mervyn Archdall. He did not obtain a permanent appointment to the honorary staff of the hospital. In later years he was glad of this; for in medical journalism he found the most satisfactory field of work. He wished for no other. Nevertheless, if the judgement of a fourth year student is of any value, he would have made a good surgeon—possibly a leader—if his health allowed. However, the profession in Australia can regard itself as fortunate that he should choose to be a leader in another sphere.

Of his qualities as a medical journalist there is no need for me to make further mention. I shall restrict myself to saying a few things about his qualities as a man. He used to say that *"De mortuis nil nisi* 'bunkum' " was a motto to which he did not subscribe. I shall avoid bunkum here.

When Archdall took over the editorial chair he felt the need of someone to help him from time to time and to look after the Journal whenever he was absent. Over a period of a few months he gave me an intensive course of training. I found him to be a patient and painstaking tutor. His knowledge of English and journalism was great; but he also had a wide knowledge of the profession and great wisdom in ethics. One could always approach him with a problem in professional conduct and be sure of a sympathetic

response and a wise ruling. At the same time he was just as ready to tell you that you were wrong—and in terms that could not be misunderstood. I can remember several occasions when he disappointed me by scarcely acknowledging a piece of writing that I had produced after many hours of hard work. But this was because he was preoccupied. In due course he would give whatever praise the work deserved.

He had a broad sense of humour and a childish sense of fun. He loved parties where people pulled crackers and ate lollies and nuts and wore paper caps. He and his staff would go to considerable pains to draw up silly programmes and to produce childish prizes for the guests at the editorial Christmas parties. From this it might be gathered that he had no time for prigs and snobs.

One aspect of Mervyn Archdall's life I have never heard mentioned by anyone but himself. It is safe to say that very few people know of it. When he was at Sydney Grammar School he wanted to row; but he was told that he was not strong enough. So he joined the Sydney Rowing Club, and he had the pleasure of rowing in a four that beat the "Grammar" crew of that day. The unrevealed sense of triumph and achievement that he experienced then recurred many times in more serious fields of endeavour. Those of us who knew him well will recognize in this one of the important traits that went to make up his complex character.

I believe Mervyn Archdall to have been a great man, with the great man's strength and weaknesses. I believe that his influence in the profession in Australia will be felt for a generation or more. As to the man himself, he was kind and thoughtful, hard when harshness was merited, frequently happy, sometimes depressed, usually patient, sometimes either amusingly or terrifyingly irascible, and in his leisure moments mischievous and playful, and always lovable.

DR. W. F. SIMMONS writes: Mervyn Archdall was an unique personality. His friends were legion, for he loved travel both within Australia and overseas and made friends wherever he went. His personal mail bore witness to this fact. His loyalty to his friends when misfortune or injustice overwhelmed them showed what manner of man he was.

He hated petty meanness and cheap publicity in the profession, but often championed the righteous cause of a hopeless minority. He was jealous of the good name of his beloved MEDICAL JOURNAL OF AUSTRALIA and gave much of what should have been his leisure time to raise its standing among world medical journals. He liked good food, expertly cooked and daintily served and shared with congenial companions, but was probably happiest entertaining a couple of his old friends in his home at a meal prepared by his wife. His tastes were simple, and as became a man whose life was dogged by unpredictable ill health, he loved good books and good music. In his younger days he had a first-class tenor voice and was a cathedral chorister.

For those of us who saw him very frequently his passing has left a gap which can never be filled. His personality seemed to unconsciously influence all those who came in contact with him; and while he would be the first to admit any fault or failing in himself, we loved him for the man he was.

Farewell, my friend.

DR. J. P. MAJOR writes: It will fall to the lot of others to write of the life and achievements of our former Editor, the late Dr. Mervyn Archdall. That he was a great editor in the medical field is beyond all doubt, and due mainly to his efforts THE MEDICAL JOURNAL OF AUSTRALIA enjoys a high reputation throughout the medical world. But, in addition to his literary attainments, his wide general knowledge and wisdom, those who were fortunate to know Mervyn Archdall well personally could not but be impressed by his innate honesty, his sincerity, his fine ideals, his sense of high purpose and his great courage in fulfilling the many important editorial duties despite very many years of physical ill health. All in all, he was a great man. I regret deeply the passing of one for whom I had such esteem and affection, and whom I was happy to regard, and to be regarded by, as a personal friend.

DR. ALAN LEE writes: Dr. Mervyn Archdall was one of the great men with whom I have had the honour of friendship. At meetings of the Federal Council, and the Board of the Australasian Medical Publishing Company Limited, and as an occasional contributor to the Journal, we had many contacts.

One of his abiding characteristics was kindliness. Many a paper accepted by the Journal owed as much to his skilful editing as it did to its author, and when I proposed to publish a book, he made the spontaneous offer to proof-read it, which I need hardly say was gratefully accepted.

At Board meetings he often seemed uncomfortable, but this was not at all due to doubts about his own position, but entirely to his fears of the unpredictability of the actions of his directors. A characteristic pose as many of us will remember him was at Federal Council meetings, when he sat at his little table, apparently dozing, sometimes not putting pencil to paper for hours at a stretch, yet always ready to speak when called upon, and able later to publish a completely accurate report of the meeting.

Though he was usually a listener, he could be quite an orator, as was shown on the occasion when his portrait was presented to him in 1955. But above all else he was a great editor, devoted to his Journal, and adamant in the maintenance of high standards of presentation and scholarship. On occasions, sitting with him in his book-lined sanctum, when his red ink markings covered too thickly one of my proffered papers, I have been provoked to argue with him, but not even the authority of Fowler would divert him from the well of English undefiled as he understood it.

THE MEDICAL JOURNAL OF AUSTRALIA is his true memorial, and I know that he was quite happy that his reputation should stand or fall with it.

DR. W. E. L. H. CROWTHER writes: Rather more than a quarter of a century ago I first met Dr. Archdall, when he was making perhaps his initial visit as Editor to Hobart. On the next occasion (he had been in ill health) he stayed at our home, and so my wife and I came to be admitted to a close and happy personal intimacy that was to continue to the end. An invitation to act as representative of the Editor in Tasmania was some years later followed by election to the Board of Directors of the Australasian Medical Publishing Company Limited. In these two capacities and as a member for five years of the Federal Council of the Association, I had an unequalled opportunity to assess the remarkable influence he exercised both from the editorial chair and in Council, on matters affecting the interests of the medical profession and the community as a whole.

How many of our members, who for year after year may have read the admirable and detailed reports in the Journal of meetings of the Federal Council, realize what calls they made on the endurance of the Editor? Sessions morning, afternoon and at times in the evening as well, for three, four or even five days, all the time at call from the President for advice, suggestions or explanation and at last back to his room at the hotel to work his notes up for immediate publication.

It was much the same, though on a larger scale, at the Biennial Medical Congress of the Association. Or again, who knew of the long hours of concentration and how much wisdom and experience were required for the week's editorial, always prepared at his home during the week-end?

Yet all these years, I do not remember him delegating such duties, in spite of increasing ill health and ever-present dyspnœa, until the Congress at Sydney two years ago, where at last he was able to enjoy a meeting without responsibility.

For myself Sydney will always mean "Grantchester", Shell Cove Road, where year after year I used to stay at their home. His wife Mary was a Tasmanian—her father Canon E. M. Thompson, Rector of St. John's Church, Hobart, and in his spare hours one of Australia's outstanding entomologists.

It was a most happy second home to me, where on the veranda overlooking Neutral Bay and the jacarandas between the road and Shell Cove, some of my happiest hours were passed. They loved to entertain, and I am sure Sir Henry Newland and medical friends from all the States will recall their dinner gatherings with tender regrets. So informal and such pleasant conversation and graced by Molly Archdall with her sister Nellie and Miss Isobel Irvine, her friend—and Mervyn in his chair—the soul of hospitality—seeing that the glasses were kept filled and smiling gently to himself as he watched over the pleasure of his friends—Mervyn Archdall indeed, and at his best and happiest.

DR. COLIN ANDERSON writes: Mervyn Archdall had a personality which I appreciated as the Honorary Secretary of a Branch and then as a member of Federal Council. During a period of four years, he gave excellent counsel, and this came from a devotion to the advancement of medicine in Australasia. One admired his philosophy and belief that we would carry on the best traditions of medicine.

Mervyn Archdall.

MR. J. H. NOLDT, Manager of the Australasian Medical Publishing Company Limited, writes: My association of more than a quarter of a century with Mervyn Archdall came to a sad close when he slipped away quietly on Friday night, September 6, 1957. I first met him when I was young and inexperienced, and shortly after he had been appointed Editor of THE MEDICAL JOURNAL OF AUSTRALIA. His influence played a major part in my career, as it did in the lives of many. As the years passed, there came a realization of some of the facets of his many-sided character, and an awareness of his love to arrange things and people. These, added to his understanding of human nature and its reactions to given circumstances and promptings, and his journalistic ability, helped him to edit and guide so successfully the Journal that was his life's work. In his early years with the Journal he was mellowed by the collaboration of such men as Henry William Armit, William Nathanial Robertson, Thomas Walter Lipscomb, David Gifford Croll, Robert Henry Todd and Richard Herbert Fetherston. They helped to develop the qualities of the man whose destiny it was to elevate the Journal to its position amongst the foremost publications of its kind throughout the world.

MISS DOROTHY TREMLETT, Lay Assistant Editor of THE MEDICAL JOURNAL OF AUSTRALIA, writes: To work for long years with a great man is an unforgettable experience. When such a man combines greatness with the capacity to inspire devotion, the experience is priceless. Such a man was Mervyn Archdall. He was my Chief for twenty years, and he taught me many things besides the technicalities of medical editing. It is possible that he did his best work during the years of the second World War, when those upon whom he had been able to rely for help with the editorial writing were nearly all in the Armed Forces or serving in other capacities. Save for one or two stalwarts, who for one reason or another were prevented from undertaking active service, he had to rely entirely on himself. Throughout those years he and I were the only two permanent members of the editorial staff. He himself has said that the experience of that time was "unique". Editorial visits to other States were naturally reduced in number, but some were made. Moreover, the whole period was punctuated by episodes of illness, sometimes serious, sometimes not so serious, but always irksome. Unless he was so ill as to be completely incapacitated, he insisted on carrying on with his work. (Here let it be said that what would have incapacitated most other men he regarded lightly.) Quite often this meant that the editorial department of the Journal was run by telephone. If he could not carry on, Dr. Calov came in as Acting Editor. Of necessity much work had to be done in the Chief's own home, and it was here that the serene and gracious presence of his wife played so great a part in his ability to keep on working. She accepted with equanimity the constant irruptions of the members of his staff, and never failed to provide them with a welcome and with delectable refreshment. In the office we worked hard—at times it seemed much too hard, especially when, as often happened, the Chief refused to take note of obstacles and wanted some very exacting task completed in an impossibly short time. But as he was always prepared to work twice as hard as anybody else, somehow the job was done. The heavy burden was always lightened by his impish (and, be it said, sometimes disconcerting) sense of fun, which led him to play pranks, like a school-boy. In the work of editing the Journal he always insisted on minute attention to detail, and on this count was not infrequently accused of pedantry. This accusation he liked to answer in the words of his own chief, the late H. W. Armit: "If by pedantry you mean meticulous accuracy, I must plead guilty." Throughout the war years Dr. Robert Scot Skirving, although he was in his eighties, was giving clinical talks to students at Royal Prince Alfred Hospital. Between him and the Chief there was a strong bond of mutual affection and respect. Dr. Scot Skirving was always a keen and interested critic of the Journal, and never failed to telephone the Chief if something particularly caught his attention. The conversation was usually punctuated by gusts of laughter from the Editor, and not infrequently ended with some such words as these: "Archdall, I must go. A couple of rats have just walked into my trap. Good-bye."

As the years went by, the permanent staff of the editorial department naturally had to be increased, and little by little the Chief began to relinquish the reins. This he found difficult to do; he frequently said that he wanted to die in harness, and it is almost what he did. Throughout the long and trying time preceding his death, his faith, always strong, continued to increase in strength, and upheld him. He always maintained that he could never have working with him any whom he could not regard as his friends. This generous attitude brought its reward. The friends he has left behind in the editorial office will never forget him, and for a long time to come it is certain that one at least of his former colleagues, when confronted with some new editorial situation, will automatically think: "What would the Chief say to this?"

DR. H. C. COLVILLE, President of the Federal Council of the British Medical Association in Australia, writes: On behalf of the Federal Council of the British Medical Association in Australia I would like to pay a tribute to the valuable services of the late Mervyn Archdall to the Council during the years that he occupied the position of Editor of THE MEDICAL JOURNAL OF AUSTRALIA. Those who have read the excellent reports of the meetings of the Council in the Journal may not have realized that their compilation was the personal effort of Dr. Archdall and involved his attendance throughout every Council meeting. It is no mean feat for anyone to get through meetings which have usually lasted for four days or more, and I can think of no greater test of endurance than to listen to a succession of speakers day after day without taking any part in the discussions. On many occasions, however, he was referred to for information and advice, and it was a tremendous advantage to the Council to be able to draw on his wisdom and wide knowledge of the affairs of the profession at a moment's notice. His kindly presence at Council meetings will be sorely missed.

DR. J. G. HUNTER, General Secretary of the Federal Council of the British Medical Association in Australia, writes: While others deal with Mervyn Archdall's character and career, I may be allowed to describe his many services, during 35 years, to the British Medical Association.

First among these, let us remember the manner in which he edited THE MEDICAL JOURNAL OF AUSTRALIA, and the admirable influence he had upon the policy of the Association. Pointing out that the Association's prime object was the promotion of medical and allied sciences, he always insisted that the Journal must be one not merely to report the Association's activities, but to deserve world-wide recognition for the highest scientific standing. This it has received, while giving wonderful service to the Association and to medicine in Australia. For this the profession owes him its gratitude.

It was an inspiration to know Mervyn Archdall, since he stood for all that is highest in the ethics and altruism of the profession. His watchwords were duty and service, his own gain farthest from his thoughts.

Nothing gave him greater pleasure than his visits to the annual meetings of the various Branches, and of kindred bodies. He thereby did much to promote unity in the profession, so essential in these days of social change. He always took an active part in the organization of the Australasian Medical Congress, and its Executive Committees invariably sought his advice.

He was a constant attendant at meetings both of the Federal Council and, Sydney being his home town, of the New South Wales Branch Council. On all matters affecting the profession's welfare he eagerly sought the views of members of the Association's various Councils.

Along with another very distinguished leader of the profession, the late Sir Archibald Collins, he represented the British Medical Association in Australia at the second General Assembly of the World Medical Association at Geneva in 1948, and at the inaugural meeting of the British Commonwealth Medical Conference in London the same year.

Taking a lofty view of the British Medical Association's objects, he would strenuously oppose anything which tended to lower the profession's dignity and honour. Throughout his career he kept his enthusiasm for the scientific advance of medicine. To this enthusiasm was due the establishment, by the New South Wales Branch, of its annual prize for an essay on a scientific subject, and by the Federal Council, of the Monograph Fund, for publication of monographs of high standard on the medical and allied sciences—this on behalf of authors unable to afford the expense. The first of these monographs has just been published.

When the Federal Council, at its meeting in Adelaide on September 1, decided to honour him, it was unanimously agreed that the honour should be the highest the Association could confer—the Gold Medal of the British Medical Association in Australia. Those intimately associated with him knew that he would desire nothing so much as this. In a letter to the President, Dr. H. C. Colville, he said: "The award left me almost a complete wreck. I realized the magnitude of the award, and it is true that the Federal Council could not have done anything which would have

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pleased me more." The pity is that he did not live to enjoy the honour longer. He has deserved well of the profession which he loved, and of which he was an ornament.

It was a privilege to be associated with him, for just on thirty years, in the affairs of the Association. Though his passing leaves a sense of great loss, the memory of him remains. May he rest in peace.

DR. P. P. LYNCH, President of the New Zealand Branch of the British Medical Association, and DR. J. O. MERCER, Editor of the *New Zealand Medical Journal*, write: Many New Zealand doctors would wish to pay a tribute to the memory of Mervyn Archdall; his death so soon after retirement was a sad blow. THE MEDICAL JOURNAL OF AUSTRALIA has great prestige and authority in this country, and is invariably linked with the name of Mervyn Archdall. He lavished on his editorial work a quality of intellectual integrity and inflexible adherence to the highest in medicine which closely reflected his own character and personality. He visited New Zealand with The Royal Australasian College of Physicians in 1950, and we regretted that ill health prevented his attending the College meeting of 1956 in Wellington. The New Zealand Branch of the British Medical Association recognizes with deep sincerity the inspired and long-continued professional service of a great medical editor.

DR. H. A. CLEGG, Editor, *British Medical Journal*, writes: I first met Mervyn Archdall in 1938, when he attended the annual meeting of the British Medical Association in Plymouth; and ever since then we have been warm friends, though space and time have imposed their limit on frequent meetings. He was one of the most generous-hearted of men I have known, and so wholesomely free from the petty envies and jealousies that mar the happiness of many who achieve eminence in their profession. Mervyn Archdall was a happy man. That was the first thing about him that struck one. This happiness was not an expression of exuberant health. It came, I think, from his religious faith and his faith in the better aspects of human nature, aspects he always looked for, and found, in those with whom he had professional and personal contact. Emerson said that an institution was the lengthened shadow of one man. THE MEDICAL JOURNAL OF AUSTRALIA certainly became an expression of the personality of Mervyn Archdall, as any journal must so become if the editor puts his heart and soul into his work. The result was seen in the growing excellence of the Journal over which he presided during the long years of his editorship. He had a keen eye for the details of his craft, and was always ready to chuckle over some of his foibles, like the spelling of the word thyroid. He was, it is hardly necessary to say in these columns, a faithful custodian of the English language, and in the leading articles and annotations set and maintained a high standard of literary excellence. But the enduring memory I shall always have of Mervyn Archdall is that of a warm-hearted and good man who greatly enriched the lives of others by his friendship.

DR. ARTHUR D'OMBRAIN sends the following tribute:

To "M.A.".

Initials of a man I seldom met
Yet whom I seemed to meet each day,
A sense of more than ordinary regret
Is mine, now you have said *Vale*,
M.A.

You stood for gracious conduct, honour's code,
All things that all of us would pray
Were said of us. You showed the open road
To true endeavour, true man's way,
M.A.

You played a leader's innings to its end
With upright bat, whate'er the play;
A man's brave heart, a kindly smile, a friend:
These things your name will always say,
M.A.

DR. CLIFFORD JUNGFER writes: On November 14, 1956, Dr. Archdall spent his last day in South Australia. He was on his way back from Perth by ship. I met him that morning, by chance, in the British Medical Association rooms. As he was free it was soon arranged that he spend the day with Dr. H. M. Saxby and myself, in the Adelaide Hills. It was a glorious day in early summer and Dr. Archdall was at his best. On arriving in Lobethal he asked to be taken inside the old Lutheran church. And, sitting there, he told us that he remembered, as a very young child, hearing his grandfather preach in Berlin. He was trying to recapture those days, and it was his calm acceptance of what life had meant for him, and of the little which lay ahead, which gave a sense of peace to us all.

When leaving the church he wrote in the visitors' book a simple phrase which expressed so well his steadfast faith and gratitude for all that life had given him: *"Gott sei dank."*

MR. L. HART writes on behalf of the printing staff of the Australasian Medical Publishing Company Limited: The staff of The Printing House has experienced a deep sense of loss in the death of Dr. Mervyn Archdall. To those of us who had been associated with him in the production of the Journal since his period as Assistant Editor his passing was felt the more poignantly, almost as a personal loss, for over the years we had come to know what manner of man he was and had learned to respect his ideals and his aspirations. It had been our privilege to introduce him to the technical details of journal production, and he displayed an intense interest in the intricate operations of printing and the way in which type could be "juggled" to fit a given space. Of the assistance given him in this regard he in after years frequently expressed generous acknowledgement. When the sudden death of Dr. Armit threw the whole responsibility for the Journal on his shoulders, the difficulties he was up against were nowhere more keenly appreciated than among those who were in daily contact with him; but we were confident that his capacity for hard work and perseverance would overcome all obstacles. As Editor, Dr. Archdall was determined at all costs to preserve the high standards of both journalism and printing set by his late chief, and that he succeeded in accomplishing this no one will doubt. Shortly after his appointment as Editor he was called on to face formidable difficulties and problems brought about by the financial depression; and it can truly be said that in that crisis, when some compromise, some easing of standards, possibly made for survival, it was characteristic of Dr. Archdall that he held steadfastly to the high principles and sound policy he had inherited.

Our late Editor's relations with his staff were at all times friendly and cooperative. He was every ready with help and advice in the many problems that arise daily in a publishing house; in particular his assistance in linguistic difficulties was frequently sought and cheerfully given, while many of us have reason to be grateful for his sympathetic and sound counsel in problems of a personal nature. A cheery and entertaining companion and an excellent raconteur, he liked to relieve the tedium of the daily routine with "the latest" told in his own inimitable fashion. He was in truth a very human and kindly gentleman, whose ambitions and achievements became a part of our daily experience; and we share in the sense of bereavement felt by all who witnessed his great service through the Journal to medicine and to medical journalism in Australia.

FRANK RAYMOND CAWTHORN.

WE are indebted to Dr. Franklyn Stonham for the following appreciation of the late Dr. Frank Raymond Cawthorn.

Dr. Frank Raymond Cawthorn died on March 31, 1957. He was born in Melbourne on July 7, 1908, and graduated M.B., B.S. of the University of Melbourne in 1931. He served for one year as a resident medical officer at the Royal Melbourne Hospital, and then proceeded to England, where he joined the Indian Medical Service. During preliminary training at the Royal Army Medical Corps College at Milbank he won the prize in hygiene. He served in various military hospitals at Quetta and other places in India, and later he was transferred to the civil branch of the service and was civil surgeon and medical officer of health, Zhob, and agency surgeon and medical officer of health, Kurram Valley. He was trained as a specialist in radiology, and had the distinction of being one of the few medical officers who attended the Staff College at Quetta and of graduating "P.S.C.". On the outbreak of war he was recalled to military service, and he commanded the 52nd Combined Indian General Hospital of 1000 beds in Burma, and later became Assistant Director of Medical Services of a division. His highly competent administration and cheerfulness, energy and devotion to duty during a prolonged period of intense strain resulted in his being twice mentioned in dispatches, and he was awarded the O.B.E. He was compulsorily retired from the Indian Medical Service on the partition of India and returned to Australia, and at first took up duty as a medical officer in the Commonwealth Department of Health. He then went into private practice at Singapore, but returned to Australia, and went into private practice with a medical group at Ferntree Gully, Victoria.

The strain of long-continued service in tropical regions left a debt against his health which later failed, and an attack of coronary occlusion forced him to abandon the rigours of private practice in a mountain area. He was appointed medical superintendent of the Mooroopna Base

Hospital, where his energy, skilful administration and cheerfulness as usual won him many friends. His sudden collapse and death during a hospital meeting terminated a versatile and colourful career. In former years he had been a prominent all-round athlete and had represented his university with success as a boxer. The deepest sympathy of all his former colleagues and friends is extended to his widow and two sons—Peter, aged 18, and Alastair, aged 15.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on March 23, 1957, at the Hamilton and District Base Hospital, Hamilton. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Lupus Erythematosus.

DR. C. E. SAWREY (Hamilton) showed a female patient, aged 22 years, suffering from *lupus erythematosus*. She had been admitted to hospital on August 6, 1956, with a one-week history of transient stiffness in various joints—wrists, hands, neck and knees—but no other complaints. Clinical examination revealed no abnormality except for some pain on movement of the joints and a little swelling. She was regarded as suffering from rheumatic fever, and was treated with penicillin and with "Disprin" (two tablets every four hours). Various investigations were carried out. A blood examination gave the following information: the erythrocyte sedimentation rate was 40 millimetres in one hour, and the leucocytes numbered 4700 per cubic millimetre, 56% being neutrophils, 1% eosinophils, 33% lymphocytes and 5% monocytes. The antistreptolysin titre was 200 units, a blood culture produced a negative result, an agglutination test for Brucella produced a negative finding, and an X-ray examination of the chest revealed appearances within normal limits.

On August 11 a butterfly rash was observed on the patient's face, and the suspicion of disseminated *lupus erythematosus* was aroused. On August 20 the Mantoux test with 1/1000 old tuberculin produced a negative result. On the same day, during the counting of 500 polymorphs in defibrinated blood, 82 L.E. cells, rosettes and occasional tart cells were seen. Microscopic examination of the urine revealed no pathological elements. Electrophoresis of the serum proteins gave the following results (grammes *per centum*): albumin 3·4, globulin 4·4, total 7·8.

On August 20 treatment with cortisone was begun, 200 milligrammes being given daily for two days, and then 150 milligrammes daily. When the dosage was reduced to 137·5 milligrammes per day the patient had a recurrence of joint pain and swelling. From September 1, chloroquine in a dosage of 750 milligrammes per day was added to the cortisone, the dosage of which was reduced to 125 milligrammes per day. This treatment produced clinical improvement and disappearance of the butterfly rash. However, laboratory tests showed little change. On examination of defibrinated blood, 41 L.E. cells were counted per 500 neutrophils. The erythrocyte sedimentation rate was 33 millimetres in one hour, and the leucocytes numbered 3600 per cubic centimetre. The patient was discharged from hospital taking chloroquine and cortisone.

Early in October, liver and renal biopsy was carried out at the Royal Melbourne Hospital, and no abnormalities were found. The administration of chloroquine was stopped, and maintenance therapy with cortisone was continued. In December this was changed to prednisolone ("Deltastab") in a dosage of 20 milligrammes per day. The patient's condition was reviewed again early in 1937. She was still complaining of transient joint pains and stiffness and of some pleuritic pain, so that the process had to be considered still active.

DR. J. L. FREW (Melbourne) said that *lupus erythematosus* had previously been thought to be a disease confined to women, particularly in the child-bearing age, but it appeared now to affect both sexes. Possibly it had previously not been recognized. There was some circumstantial evidence in support of the theory that the disease might be connected with some of the drugs now employed. Dr. Frew thought that Dr. Sawrey's treatment of the patient under discussion was correct. One of the problems concerned the reduction of the dosage. The idea was to control the disease and then reduce the dosage. Dr. Frew was of the opinion that it was best to hit hard at the start, and then lessen the dosage. The patient would need to continue treatment until she was normal and healthy again; on the dosage she was receiving, that could be four or five years. It was known that, in the treatment of long-term conditions, if relapses were permitted to occur, there was often much greater difficulty in controlling them.

DR. R. R. SOBEY (Warrnambool) asked whether there was any particular reason why ACTH should be used rather than cortisone.

PROFESSOR R. R. H. LOVELL (Melbourne) said that he agreed with Dr. Frew's statement about the difficulty of controlling the condition if relapses were allowed to occur. A review of some 200 cases of *lupus erythematosus* had been made, and on analysis the impression was gained that the response to ACTH was not always determinable. Professor Lovell believed that one might control the condition to begin with and yet still lose control. In some cases relapse occurred, and was blamed on the high dosage used.

DR. A. KERTESZ (Hamilton) said that most people used Blackburn's method of diagnosis on the proportion of L.E. cells found. The majority of pathologists would not accept as diagnostic the finding of a few L.E. cells, because such cells could be found in a number of other conditions. For the diagnosis of *lupus erythematosus* to be made, 30 or more L.E. cells must be present in the 500 polymorphs counted.

Dr. Frew said that in many cases evidence of disseminated *lupus erythematosus* was found from the start. That occurred more and more frequently, and one could not help feeling that it might be due to repeated exhibition of drugs, as he had mentioned before.

Dr. Sawrey, in reply to Dr. Sobey, said that the patient had been given cortisone rather than ACTH for the reason that in long-term conditions it was preferable to give treatment by mouth rather than by injection.

Thrombocytopenic Purpura.

DR. I. H. ANDERSON and DR. A. KERTESZ (Hamilton) showed a married woman, aged 44 years, who had been admitted to hospital on September 18, 1956, suffering from blackouts preceded by buzzing in the head and followed by hæmatemesis. She had always been a bleeder, and had bruised easily all her life. She had suffered from a perinephric abscess 38 years previously, had undergone an appendicectomy 28 years previously, and had had menorrhagia for which ovariotomy had been performed 10 years previously. Two years prior to her admission to hospital she had suffered from transient paraplegia of four days' duration. One year before her admission she had had a retro-orbital hæmorrhage behind her left eye, which had been followed by removal of the eye.

On examination, the patient was pale and obese; bruising and petechiæ were noted on the limbs and trunk, and hæmatemesis was present. No viscus was palpable in the abdomen. A full blood examination was performed; the hæmoglobin value was 13·5 grammes *per centum* (91%), the leucocytes were normal and no abnormality was detected in a blood film; the platelets numbered 31,000 per cubic millimetre (normal, 250,000 to 400,000) and were of queer shape. Marrow aspiration revealed normal erythropoiesis and myelopoiesis; the megakaryocytic series was increased and there was partial maturation arrest with poor granularity; platelet production was of giant type.

In the absence of any ætiological factors in the patient's history, a diagnosis of idiopathic thrombocytopenic purpura was made, and the long-acting type of ACTH was given in a dosage of 100 milligrammes per day, with delta-hydrocortisone, five milligrammes every six hours. After a further hæmatemesis the patient was given a transfusion of two pints of fresh blood. The platelet response to those measures was poor; after five days the count was 53,000 per cubic millimetre, and epistaxis and hæmatemesis were still occurring to a slight extent. It was therefore decided to remove her spleen. That procedure presented no technical difficulties, and the post-operative period was uneventful.

The day after operation the platelet count had risen to 105,000 per cubic millimetre, and no new hæmatemesis or bruising had appeared. The dosage of ACTH was reduced by 25 milligrammes per day until the fourth day after operation, when the administration of prednisolone and ACTH was discontinued to favour healing. For 14 days after operation she progressed well, and the number of platelets rose to 170,000 per cubic millimetre. On the fifteenth day, 11 days after the cessation of ACTH depot therapy, she had an epistaxis, and the platelet count was 41,000 per cubic millimetre. Treatment with hydrocortisone, five milligrammes every six hours, was begun, and the number of platelets rose slowly to 89,000 per cubic millimetre over the next 14 days, during which period she was free from symptoms and signs. In the succeeding months the dosage of hydrocortisone had been progressively reduced to five milligrammes every other day at the time of the meeting. The platelet count was consistently between 110,000 and 95,000 per cubic millimetre. The patient was free from symptoms, no longer bled abnormally when she cut herself and had no abnormal bruising tendency or epistaxis. Her hæmoglobin value was 14·5 grammes *per centum* (100%).

Professor Lovell, in answer to a question by Dr. Anderson, said that perhaps the initial dosage of cortisone had been rather small; but on the other hand, 100 milligrammes per day was a fair trial. When homœopathic dosages were used, the problem was whether one should stop or give more. He himself felt that it was better to try to stop. At the time of the meeting, the patient was receiving what actually amounted to enough to put her own adrenals to sleep, as it was only replacing what she herself produced, instead of encouraging her own adrenals to produce sufficient, although she might remain well. Professor Lovell thought it advisable always to try to avoid giving small continuous doses, since to do so might lessen resistance to any infection that occurred.

Dr. Frew said that the case presented a problem in diagnosis; he did not think that Dr. Anderson was out of the wood. In his own experience the so-called idiopathic purpura was something quite different. One patient, who after 15 months had been diagnosed as suffering from leuchæmia, had a giant type of megakaryocyte which to some extent was circulating in the blood. Dr. Frew stressed the importance of not being too complacent about the diagnosis. On the question of splenectomy, he thought that a blood transfusion should be tried first. In the chronic stage a splenectomy was the method of treatment; in the acute stage cortisone was given and the spleen was left.

Dr. Kertesz, in reply to Dr. Frew's remarks about splenectomy, said that patients whose condition was advancing were the most likely to benefit from it. Otherwise ACTH should be given.

Monocytic Leuchæmia.

DR. A. KERTESZ and DR. D. SUNDARASON (Hamilton) showed a boy, aged four years, who had been admitted to hospital on June 27, 1956, with an eight days' history of progressive lassitude, pallor and irritability. On examination, he was seen to be pale; his pulse rate was 132 per minute and his temperature was 100° F. His liver was palpable one finger's breadth below the costal margin, and the spleen three fingers' breadth. Generalized lymphatic enlargement was present. A provisional diagnosis of acute leuchæmia was made. A blood examination gave the following information: the hæmoglobin value was 35% (5·2 grammes *per centum*), and the erythrocytes numbered 1,850,000 per cubic millimetre; the leucocytes numbered 2000 per cubic millimetre, and many were atypical blast forms, possibly paramyeloblasts or monoblasts. The pathological opinion was that the appearances were consistent with acute leuchæmia. Marrow from the iliac crest was examined, and the findings suggested that the condition was monoblastic leuchæmia.

The patient was treated with penicillin in a dosage of 50,000 units every six hours for seven days, and his temperature fell to normal. A transfusion of one pint of blood was given, and the hæmoglobin value rose to 76%. The administration of ACTH (five international units every six hours) was started on the third day and discontinued after ten days. Cortisone treatment was then immediately begun, in a dosage of 25 milligrammes three tims a day until the patient's discharge from hospital. "Puri-Nethol" (6-mercaptopurine) was administered from the fourth day, the dosage being 50 milligrammes per day. About two and a half weeks after the patient's admission to hospital a rise in temperature occurred, and for that reason "Achromycin" was given for seven days. The hæmoglobin value in the fourth week was 34%. The patient was given a transfusion of one pint of blood, and discharged from hospital three days later (July 27) taking 50 milligrammes of "Puri-Nethol" per day.

Serial blood examinations showed that a blood picture near enough to normal was being maintained. The hæmoglobin value varied from 80% to 89%, and the white cell count varied from 4200 to 6900 per cubic millimetre; on two occasions a moderate degree of eosinophilia was present. When last examined on March 18, 1957, the child looked healthy and cheerful. Cervical glands were palpable on both sides, the liver was not palpable, and the tip of the spleen was just palpable on deep inspiration. A full blood examination gave the following results: the hæmoglobin value was 79%; the leucocytes numbered 9295 per cubic millimetre, and the neutrophils showed a "shift to the right" with toxic granulation; atypical mononuclear leucocytes were present but scanty.

DR. ROBERT SOUTHBY (Melbourne) referred to the short history. He thought the condition difficult to pick on clinical investigation. There was some resemblance to the difficulty related to infected tonsils in children, which caused trouble and then cleared up; in the intervening period the child could be taken as suffering from an arthritic condition, with a high temperature and rapid heart rate, as in rheumatic fever. A full blood examination gave the answer. At the present time, with cortisone there was the possibility of relapse. However, while the improvement might not be maintained for long, the treatment was well worth trying. Dr. Southby said that the blood transfusion service had been worried by the demand for blood for leuchæmic patients when there was no ultimate response. The service had been designed to provide blood as a life-saving measure. In cases of leuchæmia it was essential to have relatives to act as donors; the ultimate outcome was that blood was provided for every patient with leuchæmia, and such donors increased the list of donors. Dr. Southby

asked whether the child under discussion had a history of sore throats. He said that the general impression was that there were more cases of leuchæmia in recent years. Referring to the length of time for which treatment should be continued, Dr. Southby said that he would be inclined to keep to the present dosage for at least another month, and if the child showed any signs of recurrence, to start again.

DR. C. MACDONALD (Hamilton) referred to the problem of the effect of X rays used on the mother during pregnancy—was it related in any way to the increase in the incidence of leuchæmia? Sir Macfarlane Burnet had implied that the connexion had been proved. Dr. Macdonald said that he and his colleagues at the Women's Hospital, Melbourne, were not satisfied that that was so. He thought that the case against X rays had not been proved. He asked whether there was any history of the use of X rays in the case under discussion.

DR. PORTER (Ballarat) said that a run of cases of infective mononucleosis was occurring in Ballarat, and he had been struck with the similarity between the findings in those cases and in the case under discussion.

DR. J. L. FREW (Melbourne) said that he thought it fair to say that for leuchæmia there was no effective treatment. Referring to the use of blood transfusions, he asked whether it was in fact a waste, because the effect was only for a short time. Was the policy of no treatment justified? Were there any cases of acute leuchæmia in which treatment was justified? Those were the responsibilities that clinicians had to face. He wondered whether there was an underlying infection. He thought that the patient under discussion had had a remission, but was still suffering from the disease and would probably relapse. The other question was whether blood transfusions would need to be given (and would be followed by remissions for perhaps three, six or twelve months), and then the whole process would have to be repeated. Dr. Frew thought that if the condition was monocytic leuchæmia, the treatment was justified.

DR. W. R. ANGUS (Warrnambool), referring to the high incidence of blood diseases in the Hamilton area, said that there was a pathologist there. If there was a pathologist in Warrnambool, there would possibly appear to be an increase in blood disease there. Dr. Angus recalled one patient suffering from leuchæmia who had died within seven days after treating himself with sulphonamides. In a few years probably more blood diseases would be seen. Dr. Angus thought that if research could be done on the effects of sulphonamides given to pregnant women, they would be found to have more bearing on the subject than X rays.

Dr. Kertesz, in reply to what had been said in discussion about the use of blood transfusion in monocytic leuchæmia, said that he would not be prepared to withhold it. He tried to bring the patient's hæmoglobin value back to 12 grammes *per centum*. He did not think that the use of blood transfusion in leuchæmia should be regarded as a special case; it was a recognized method of treatment. Dr. Kertesz could not say whether there was an increased incidence of leuchæmia; in England it was thought that there was an increased incidence, due to more accurate diagnosis. With regard to the onset, Dr. Kertesz said that when monocytic leuchæmia was suspected, he always asked whether the patient had had a sore throat. In the case under discussion there had been only a history of a cold, and he was surprised at the result of treatment. The history was usually difficult. Anæmia was generally a feature. In blood films he had seen that the erythrocytes were comparatively normal, and he regarded them as particularly important. He suggested that if patients were examined in time, they could be treated effectively.

Myasthenia Gravis.

DR. H. JOHNSON (Hamilton) showed a married woman, aged 76 years, who had presented on November 6, 1956, having been referred by Mr. S. C. Fitzpatrick; she complained of diplopia of three weeks' duration, usually vertical and on looking downwards. It was worse in the later part of the day. She had had a transient episode of diplopia five months previously. In 1929 she had undergone thyroidectomy for toxic goitre, and she had been hypertensive for many years.

On examination of the patient, bilateral ptosis was present when the head was tilted back. The diplopia was due to extra-ocular muscle weakness not corresponding to any neurological pattern. The pupils were equal and active. Examination of the optic fundi revealed hypertensive and arteriosclerotic changes in the retinal vessels. No abnormality was detected in the cranial nerves. No wasting had occurred. An injection of atropin (1/150) followed in 15 minutes by 2·5 milligrammes of "Prostigmin" produced restoration of binocular vision and resolution of the ptosis, with slight residual weakness of the action of the right inferior oblique muscle. General examination gave no relevant information. The blood pressure was 206/108 millimetres of mercury. The urine was normal. An X-ray examination of the chest revealed senile emphysema and an increased transverse cardiac diameter. There was no evidence of thymoma.

The patient was treated with rest. Her condition was fairly well controlled with "Prostigmin", one tablet on rising, one at 9 a.m., two at midday, two at 4 p.m. and one at 8 p.m. She regulated her own dosage. Tincture of belladonna was given 20 minutes before the tablets, otherwise she complained of abdominal pain. A change had later been made to "Mestinon", one tablet every four hours throughout the 24 hours; the patient preferred "Mestinon" to "Prostigmin". Potassium chloride was given with each tablet.

Bulbar involvement had subsequently occurred, and the patient became fatigued on chewing and swallowing and had difficulty in speech. Neck weakness made her head fall forwards. The limb muscles had remained unaffected, although diplopia at times made walking uncertain. She had had no attacks of dyspnœa.

PROFESSOR R. R. H. LOVELL (Melbourne) said that some years earlier *myasthenia gravis* had been thought to resemble curare poisoning. There appeared to be no particular method of treatment but to use the right amount at the right time. It was a disease in which there were little collections of small lymphocytes between the muscle fibres. Attention had been drawn to them again in the U.S.A. in about 1947, in cases of rheumatoid arthritis.

DR. J. L. FREW (Melbourne) said that patients with *myasthenia gravis* took "Prostigmin" very well. In the crises during investigation, or when a surgical condition of the thymus was present, it was difficult to know whether enough was being given. Myasthenia in a mild form was fairly common, and became an urgent problem when infection occurred. On biochemical grounds he thought that if such patients had an overwhelming infection, they might present an emergency requiring urgent treatment.

MR. S. C. FITZPATRICK (Hamilton), referring to the treatment of the patient under discussion, said that an ordinary dose of "Prostigmin" (15 milligrammes) gave her pain in the stomach, and so tincture of belladonna was given with each dose. "Mestinon" had been more helpful, as the belladonna, while preventing pain, made the patient's mouth dry. She had required no belladonna while taking "Mestinon" tablets.

Intrabiliary Rupture of a Hydatid Cyst in a Child.

DR. C. E. SAWREY and MR. S. C. FITZPATRICK (Hamilton) showed a girl, aged 10 years, who had been admitted to hospital on March 23, 1956, with severe spasms of abdominal pain of one week's duration. The pain was generalized over the upper part of the abdomen, and varied in intensity. She had anorexia, vomiting and constipation, having had two bowel actions in the past week. She had no cough, and had not lost weight recently. Investigation of her past history revealed that she had fallen off a truck and bruised her abdomen on November 19, 1954, and had been in hospital for 48 hours. Her past illnesses included mumps, measles, whooping-cough, chickenpox, scarlet fever and poliomyelitis. Her tonsils and adenoids had been removed, and secondary hæmorrhage had occurred. At the age of seven months she had undergone an operation for intussusception. She had one sister, a spastic.

On examination of the child, she was pallid, miserable and sunken-eyed. Her temperature was 101° F., her pulse rate was 130 per minute, and her respirations numbered 24 per minute. No abnormality was detected in the heart and lungs. Generalized tenderness and guarding were present over the abdomen. She had spasms of severe pain at intervals. The urine was alkaline, and contained no albumin and no reducing substances; acetone was present, but no diacetic acid; the specific gravity was 1020. A blood examination gave the following information: the total number of leucocytes was 16,100 per cubic millimetre, of which 81% were segmented neutrophils, 10% non-segmented neutrophils, 7% lymphocytes and 2% monocytes; there were no eosinophils. The neutrophils showed a "shift to the left", and the number of platelets was slightly increased. Her blood group was O(4). X-ray examination revealed slight elevation of the right hemidiaphragm; the area of liver dullness extended downwards, and there was general opacity over the whole of the upper part of the abdomen; the base of the right lung appeared clear.

After consultation with Mr. John Kneebone, medical treatment was considered indicated. Intravenous therapy with dextrose solution (4% in normal saline) was begun, and penicillin was administered. After 48 hours, the child's condition had greatly improved. She had no biliuria, and had one attack of severe abdominal pain which lasted for ten minutes. She had a high temperature for two weeks, which settled by lysis. There was no response to antibiotics. No diagnosis was reached. She was discharged from hospital on April 7.

She continued to have spasms of abdominal pain, lasting for 10 minutes, about once a week, and once jaundice was present. On August 28, 1956, she was readmitted to hospital for investigation. Her general condition was good. The outline of liver dullness was raised in the axillary line well above normal, but there was no enlargement below the costal margin. A blood examination gave the following information: the total number of leucocytes was 20,900 per cubic millimetre, 47% being segmented neutrophils, 3% non-segmented neutrophils, 5% eosinophils, 2% basophils, 37% lymphocytes and 6% monocytes; the erythrocytes and platelets were normal. The most notable abnormality found on X-ray examination was an immobile domed elevation of the right hemidiaphragm, over which the pulmonary trunks appeared to be spread. Above it an interlobar effusion could be seen in the lesser fissure. It was difficult to locate the upper level of the liver, which did not appear to be enlarged downwards. The impression was gained that the pulmonary changes were secondary to subdiaphragmatic disease; the most likely disorders were thought to be a hydatid cyst on the upper surface of the liver, a hepatoma or a subphrenic collection of fluid which had become walled off. The prepyloric portion of the stomach was much narrowed and appeared rigid, and the duodenal cap was deformed and puckered. That raised the possibility that the acute upper abdominal episode which had occurred some months previously might have been due to leakage from there, with a subsequent subphrenic collection. It was thought possible that rupture of a hydatid cyst in the *porta hepatis* region could produce the same picture. The Casoni intradermal test produced a strongly positive reaction.

When the course of the illness, the increase in the number of eosinophile cells, the strongly positive response to the Casoni test and the progressive changes in the radiological findings at the dome of the right hemidiaphragm were taken into account, it was concluded that there was no doubt about the diagnosis of subdiaphragmatic hydatid cyst with secondary involvement of the right lung pleura and lower lobe. Preparatory treatment for operation was therefore begun on September 10. Operation was undertaken on September 19 by Mr. S. C. Fitzpatrick assisted by Mr. A. D. Matheson. Under "Pentothal", nitrous oxide and oxygen anæsthesia (Dr. M. C. Piercy), right thoracotomy was performed, and through a transpleural approach to the right lobe of the liver the cyst was removed and the adventitial wall was closed. The combined diaphragm and adventitial wall were almost half an inch in thickness. The cyst wall had collapsed and was œdematous and stained yellow, and the contents were jelly-like in consistency; no daughter cysts were present. No pathogenic organisms were grown on attempted culture. It was concluded that the cyst had leaked into the biliary canals, and that bile had leaked into the adventitial cavity. The patient's convalescence was uneventful, and she was discharged from hospital on September 22.

MR. G. NEWMAN-MORRIS (Melbourne) said that little hydatid disease was now seen in Melbourne; that probably made the teaching incomplete, since the need to consider hydatid disease in the making of a diagnosis would not be stressed. A second point concerned a statement that had previously been made to the effect that hydatid disease was on the wane, as a result of the fact that in the country it was no longer the custom to kill meat for domestic use on properties, so most dogs were no longer fed on offal.

MR. A. D. MATHESON (Hamilton) said that education on that matter took some time.

DR. J. L. FREW (Melbourne) asked what was the rate of growth of the cyst in the case under discussion.

DR. C. F. BALDACCHINO (Merino) said that it was possible to control the giving of offal to dogs, but it was impossible to stop them from catching rabbits. That raised the question whether dogs could contract hydatid disease from rabbits. He wondered whether anyone present could answer that question.

DR. W. R. ANGUS (Warrnambool) said that in the case under discussion the cyst was probably not a solitary one. He asked whether Mr. Fitzpatrick thought there was any risk of recurrence of cysts.

Mr. Fitzpatrick said that in similar cases the diagnosis of hydatid disease was not always made because it was not always thought of. The first stage in diagnosis was suspicion. They had learnt to be suspicious of biliary colic etc.; it could be due to many things, and often it was the cause of the patient's seeking attention. In reply to the question whether hydatid disease was on the wane, Mr. Fitzpatrick said that nine new cases per year were seen in Hamilton; too many children had the disease. With regard to the rate of growth of cysts, he said that cysts in the lung grew on an average between half an inch and one inch each year. He believed that the results of the Casoni test and the complement-fixation test remained positive for many years after a cyst had been removad. One had to keep in mind the possibility that further cysts might develop later; it might be five or ten years before they appeared. Up to adolescence, negative results to the Casoni test were no use. The number of eosinophils might be considerably increased, sometimes up to 39%. In reply to Dr. Baldacchino's question about the control of hydatid disease in dogs, Mr. Fitzpatrick said that he had yet to see an example of true hydatid disease in rabbits.

Placenta Accreta Presenting as Ante-Partum Hæmorrhage.

DR. C. MACDONALD (Hamilton) showed a married woman, aged 32 years, who had presented on January 10, 1957, with a history of having lost blood and clots *per vaginam*. She was then 24 weeks pregnant, according to calculation. Her last menstrual period had occurred on July 13, 1956. She had had no previous serious illnesses or operations. The patient had six children ranging in age from twelve to three years. Post-partum hæmorrhage following the birth of the youngest child had required blood transfusion. Two years prior to examination the patient had had a miscarriage when she was 26 weeks pregnant; on that occasion she had required blood transfusions prior to the miscarriage and several pints of blood had to be given afterwards.

On examination of the patient, the condition of the fundus corresponded to the period of amenorrhœa, fœtal movements were palpable, and there were some blood clots in the vagina, but no active bleeding was occurring. The os was closed. The hæmoglobin value was 80%. The patient rested in hospital for two days and no further bleeding occurred. She was then discharged, with instructions to take five grains of ferrous gluconate three times a day.

On January 29 she returned, with a history of further severe vaginal hæmorrhage. There was no pain. She was now 28 weeks pregnant, and the height of the fundus corresponded with that stage. Fœtal movements were still palpable. Blood clot was present in the vagina, and there was a slight trickle of bright blood from the os, which was closed. The hæmoglobin value was 65%. With rest in bed the bleeding soon stopped, but the patient was given a transfusion of two pints of blood. On the following day the hæmoglobin value was 84%. She remained in bed for the next few days, and no further bleeding occurred. On February 3 clear liquor began to drain; there was no bleeding. That state continued for six days, without pain. On February 9, at 7 p.m., the patient suddenly had a brisk hæmorrhage, the estimated loss being about two pints. Some vague lower abdominal pain had been present for about two hours previously. One-quarter of a grain of morphine was given, and the transfusion of two pints of blood was begun. The pregnancy was only in its thirtieth week, and in the circumstances the hope of continuing it was remote.

Examination of the patient under anæsthesia revealed that the os was dilated to admit three or four fingers, the vertex was presenting, and what was thought to be the placenta encroached on the lower uterine segment posteriorly. An internal version was performed and a leg was brought down, in the hope that the half breech would act as a hæmostat. However, with only moderate traction the buttocks were delivered, followed by the thorax. With further traction the head slowly negotiated the cervix. A stillborn male child weighing one pound three ounces was delivered. Ergometrine (0·5 milligramme) was then given intravenously; but the placenta showed no signs of separation, and a moderate flow of blood continued. With the patient still under anæsthesia, manual removal of the placenta was undertaken. After a few minutes' exploration, it was found impossible to define a plane of cleavage between the placenta and the uterine wall. An attempt was then made to remove the placenta piecemeal, but only small lumps of cotyledons came away in the hand, and hæmorrhage continued. It was then realized that the problem was one of *placenta accreta* with post-partum hæmorrhage, so the decision was made to proceed with hysterectomy. While a further two pints of blood were being cross-typed, the patient was given a transfusion

of two pints of plasma. Subtotal hysterectomy was performed, and although at the completion of the operation her pulse rate was 130 per minute and her blood pressure was 90/60 millimetres of mercury, when the blood transfusion was given her condition rapidly improved. Her convalescence, apart from slight infection at the lower end of the wound, was uneventful. Twenty-four hours after operation the hæmoglobin value was 80%. In all she had received four pints of blood and two pints of plasma.

DR. W. IVON HAYES (Melbourne) said that the title of the case was misleading, since in a total *placenta accreta* there was no bleeding. On clinical examination of the patient after delivery, the uterus did not contract; it was flabby, but there was no hæmorrhage. Hysterectomy was required, otherwise hæmorrhage was inevitable. The basic reason was not the line of cleavage. Partial *placenta accreta* in which the cleavage was not in one particular area was very common; it was a complication that produced post-partum hæmorrhage. After delivery, examination might show that the uterus contracted, but not in one spot. There was a soft spot perhaps the size of a shilling. The treatment was then by brute force—tearing off. With blood transfusion the problem was overcome. If the placenta could not be dislodged, it would be found to be adherent at the soft spot. In a case of *placenta accreta*, the pulling down of a leg was excellent treatment.

DR. G. SIMPSON (Melbourne) said that Dr. Macdonald was fortunate, in that the patient under discussion had other children; otherwise he might have had a problem. Dr. Macdonald had realized that he was dealing with a partial *placenta accreta*. Apparently blood for transfusion was not so readily available in the Hamilton area as in Melbourne; in Melbourne more blood was given, and in the majority of cases the bleeding was stopped. That should be kept in mind, in that by early diagnosis it was possible to manage with a transfusion of four pints instead of 14. In the case under discussion, trouble could have arisen if hysterectomy had not been performed. Dr. Simpson said that one of the most dramatic influences in obstetrics in recent years was "Fibrogen", which was given in such cases in the hope of controlling the bleeding. Operation in many cases was not effective if much hæmorrhage occurred, and hysterectomy had to be performed later. In cases of total *placenta accreta* the placenta could be left behind and so absorbed.

Dr. Hayes said that he agreed that if there was no bleeding, the placenta could easily be left.

DR. J. W. JOHNSTONE (Melbourne) said that in the higher forms of life the villæ actually entered the blood vessels and received blood from the mother. That assisted in the production of collections of fibrinogen which produced the line of cleavage. Dr. Johnstone asked whether the mother had previously had a curettage; he said that that was a common cause of the condition under discussion.

DR. J. G. SHELTON (Melbourne) questioned the incidence of complete *placenta accreta*, which had been given as one in 2000; he said that those were pathological figures. Eastman had recently stated that on pathological grounds one in 100 retained placentas was a partial *placenta accreta*.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (BRITISH MEDICAL ASSOCIATION).

Annual Scientific Meeting.

THE seventeenth annual scientific meeting of the Ophthalmological Society of Australia (British Medical Association) was held in Melbourne from September 30 to October 4, 1957, DR. KEVIN J. O'DAY, the President, in the chair. After a formal opening by the Lieutenant-Governor of Victoria, Sir Edmund Herring, the President opened the proceedings with a paper on the comparative anatomy of the eye. Then followed a varied programme packed into every available moment of the week.

Four overseas guest speakers were present, headed by Sir Stewart Duke-Elder, who discussed closed angle glaucoma, the handling of which has advanced recently. It is now agreed that an early prophylactic peripheral iridectomy in the second eye is indicated, provided that no signs of chronic simple glaucoma are present. Dr. Arthur Bedell (New York) showed two series of colour photographs of the fundus, demonstrating the invaluable help of this method of recording and following the progress of disease. Mr. P. Jameson Evans (Liverpool) contributed, with Dr. S. G. Anderson (Melbourne) and Mr. Frank Phillips (Hobart), to a symposium on *herpes simplex* infections of the cornea. General condemnation of the use of steroids in the presence of corneal lesions emerged from this discussion. Dr. Hedwig Kuhn (Indiana) demonstrated methods of screening used in industrial ophthalmology, to suit visual capacities to the task.

The outstanding paper of the meeting was from Professor S. Sunderland (Melbourne), on the anatomy of the nerves to the extraocular muscles, and his definitive proof of the existence of proprioceptive endings in the muscles themselves.

Mr. Roger Darvall (Sydney) provided a new and stimulating idea in a paper on the local use of androgens and œstrogens in the treatment of a variety of resistant and periodic corneal diseases. Another ray of hope was offered by Dr. Pincus Taft, Mr. J. Bignell and Dr. G. Kurrle (Melbourne) in a review of diabetes, its increasing incidence and the control of new vessel formation on the surface of the retina by small doses of X rays, thus preventing some of the vascular disasters leading to early blindness.

Mr. Arthur Joyce (Melbourne) added to his now internationally known method for the treatment of retinoblastoma by radon seed implantation.

Dr. Kate Campbell (Melbourne) added a postscript to the triumph of the control of retrolental fibroplasia by the avoidance of high oxygen concentrations in premature infants, quoting the latest figures for the very low incidence of the disease.

Professor M. Ewing (Melbourne) considered the involved question of parotid gland tumours and their relation to Sjögren's syndrome, Mickulicz disease and Hasimoto's disease.

Mr. Hugh Greer (Melbourne) contributed another valuable paper, this time on conjunctival carcinomata.

Mr. K. B. Redmond (Orange) discussed the place of microcorneal lenses in the treatment of keratoconus, and a general discussion emphasized some of the dangers of this type of lens.

The question of the education of legally blind children in New South Wales was dealt with by Mr. F. Gregory Roberts (Sydney), and considerable debate followed on the wisdom of compulsory registration of the blind. The opinion of the meeting was generally against it.

Mr. Peter English (Brisbane) added to his study of lesions of the eye staining with Bengal rose, Mr. A. Lamb (Perth) compared modern suture materials in detail with the virtues of human hair, and Mr. Geoffrey Serpell (Melbourne) discussed xanthelasma.

Cases were presented by the following: Dr. J. Billings (Melbourne), Refsum's syndrome; Dr. M. E. Cameron (Brisbane), arterio-venous aneurysm; Mr. K. C. Bradley (Melbourne), neurosurgical conditions.

These papers will be printed *in extenso* in Volume XVII of the Transactions of the Society, to appear in 1958.

The next meeting of the Society will be held in Adelaide from October 6 to 12, 1958. The President will be Dr. Michael Schneider, the Vice-President, Dr. J. L. R. Carter, the Secretary, Dr. Peter Rogers, and the Treasurer, Dr. A. E. F. Chaffer.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

HOSPITAL ABUSE.[1]

[From the *Australasian Medical Gazette*, June, 1890.]

A LARGELY ATTENDED MEETING of the Medical Society was held last month under the presidency of Mr. T. M. Girdlestone at which the consideration of the question of hospital abuse was continued. The following resolutions were discussed and finally carried unanimously:

This Society is of opinion that: 1. Great imposition on the part of well to do people is practised at the public hos-

[1] From the original in the Mitchell Library, Sydney.

pitals which is contrary to the principle on which these institutions were founded and on which they should be conducted. 2. All hospitals receiving Government aid annually should be devoted solely to the treatment of the destitute and poor. 3. Paying patients should not be received into hospitals receiving aid for the treatment of the destitute and poor. 4. A wage limit should be fixed for all hospital patients (i.e., those earning more than a certain amount should be excluded). The Society suggests a wage limit of £2 a week for single men, £3 a week for married men, and £6 a week for families in which there are several wage earners. That the circumstances of each applicant for admission should be investigated by an officer specially appointed for the purpose who shall have wide discretionary powers in special cases. It was decided to appoint Drs. Balls-Headley, Barrett, Girdelstone, Jackson, Neild, and J. P. Ryan as a committee to represent the opinions of the Society to the Charitable Commission.

Correspondence.

FLUORIDE AND DENTAL CARIES.

SIR: The letter of L. R. A. Williams on "Fluoride and Dental Caries" in THE MEDICAL JOURNAL OF AUSTRALIA, November 16, 1957, page 742, leaves little doubt that the "uncertainty surrounding some aspects" (P. Pincus, M. J. AUSTRALIA, November 16, 1957) of this topic is non-existent.

Several aspects of the letter of P. Pincus are extremely misleading, and bear no relation to the question of fluoride and dental caries. The reference to industrial toxicology has no bearing on the safety of controlled fluoridation at the levels recommended by health authorities. It has been recognized by all competent workers in the field of fluoride research, particularly since the publication of Roholm's monograph in 1937,[1] that in certain industries, workmen are exposed to excessive concentrations of fluoride which produce chronic fluoride intoxication; but even under these circumstances, the mortality and morbidity of the workers are not increased, and as the report of Agate *et alii*[2] shows, "none of the workers was found to suffer clinical disability", although 32·5% showed radiologically demonstrable bone abnormalities, a number having been exposed to high fluoride concentrations for over 50 years.

The most recent authoritative opinion on the safety of water fluoridation given by the Expert Committee of the World Health Organization,[3] September 4, 1957, after an examination of the scientific evidence, was that: "All these findings fit together in a consonant whole that constitutes a great guarantee of safety, a body of evidence without precedence in public health procedure." It is precisely because of the intensive research into the toxicology of fluoride that the limits of safety are so exactly known, and that the "effectiveness, safety and practicability of fluoridation as a means of preventing dental caries, one of the most prevalent and widespread diseases in the world, is now established".

In view of these statements, it is not surprising that the New Zealand Commission said, in its general conclusions:[4] "We are satisfied that widespread use should be made of the fluoridation process for the purpose of achieving an urgently needed improvement in the present serious state of dental health in New Zealand."

The statement that 1·0 part per million of fluoride is capable of affecting bacterial growth is not supported by the observations of other investigators, and furthermore, in a report published in 1949,[5] where P. Pincus tested the sensitivity of two Gram-negative bacilli to fluoride, "one withstood exposure to broth containing 1,000 parts per million", and also "the results indicate that fluorine *may* exercise a toxic effect on the bacteria tested, though part of the inhibition of growth may have been due to the acid pH".

The anxiety that Paul Pincus shows for the "sick Australians" is to be commended, but surely this is an exaggerated sympathy in the light of the evidence that ill effects have never been detected as a result of the consumption throughout life of drinking water containing the optimal amount of fluoride. It would appear that in the interests of improving the dental health of the community, an improvement which has been undoubtedly proved to take place, the anticipation of some nebulous ill effect should not be allowed to outweigh the evidence which has accumulated from years of research, and which has led to the endorsement and the advocacy of fluoridation by the highest health authorities in the world.

Yours, etc.,

NOEL D. MARTIN.
Associate Professor of Preventive Dentistry,
Faculty of Dentistry,
University of Sydney.
November 22, 1957.

[1] Roholm, K. (1937), "Fluorine Intoxication: A Clinical-Hygienic Study", Lewis, London.

[2] Agate, J. N., *et alii* (1949), *Medical Research Council of the Privy Council, Special Report Series No. 22*, "Industrial Fluorosis", H. M. Stationery Office.

[3] World Health Organization Press Release, WHO/45, September 4, 1957.

[4] Report of the Commission of Inquiry (1957), "Fluoridation of Public Water Supplies", Government Printer, Wellington, New Zealand, 154.

[5] Pincus, P. (1949), "Fluorine and Caries", *Brit. Dent. J.*, 87: 325.

NEUROSURGERY AT HOME AND ABROAD.

SIR: It is fortunate that in this country newspaper adulation is seldom the source of professional fame or eminence. However, during recent years the Australian public apparently has come to accept newspaper and broadcasting choice of consultants as authoritative and compelling. The much publicized and publicly subsidized trips of patients to a neurosurgical clinic in Sweden is a current example of this attitude.

Those in a position to judge such matters do not doubt the competence of Australian consultants in all branches of work. They do not lag behind world advances in ideas and techniques. Even so, man is mortal, and surgery still faces defeat at the end of many roads. Tumours may still be inoperable, and wisdom may still triumph over a callous urge to mutilating eradication.

It is a pity that such facts are not grasped by our newspaper and broadcasting philanthropists, who are too apt to assume that whatever is inoperable here will be operable in a foreign land and that the quest is worthy of public support. For it seems at present that patients in all stages of pathological dilapidation must be endowed by well-meaning subscribers and, surrounded by a publicity in which local inadequacy is necessarily implied, rushed off to Sweden.

Necessarily in each Australian capital there are but few neurosurgeons. For these reasons interstate consultations in difficult problems are worthy of consideration, and if there is an insistence that more distant centres must necessarily be better, the famous centres of Great Britain lack nothing, and the more ready use of medical reference would save many unnecessary calls on public charity. Such sensible measures would, of course, lack the tense drama of a flight to foreign lands.

Yours, etc.,

149 Macquarie Street, DOUGLAS MILLER.
Sydney,
November 22, 1957.

SKIN CANCERS.

SIR: In the November 16 issue of THE MEDICAL JOURNAL OF AUSTRALIA, David L. Dey, in discussing the management of skin cancers, stated: "I should hesitate to mention such mediæval methods as destruction by cautery or carbon dioxide snow and other similar manœuvres."

As a dermatologist who has treated thousands of skin cancers, I am obliged to disagree with him regarding the worth of cryotherapy. Cryotherapy has a very definite place and sphere of usefulness in treating certain skin malignancies, and is far from being a mediæval manœuvre. For curing such lesions as intraepidermal carcinomata and Bowen's patches on the back, it is probably the method of choice. Experienced dermatologists and radiotherapeutists know that the back does not stand radiotherapy well, and that even with sometimes relatively small doses post-radiational ulcers can develop. I have always been given to understand by surgeons that they do not like swinging "flaps" about on the back, as the "take" rate is not the best.

Another useful sphere for cryotherapy is as a preliminary treatment for squamous celled and basal celled skin

carcinomata. The bulk of the tumour can be removed by that method, and the amount of X-ray therapy subsequently needed for cure reduced. This combined method produces better cosmetic results than where only radiotherapy in higher doses has been used.

A prerequisite for cryotherapy, however, is that the operator must know how to use it. That knowledge comes with experience very largely.

Furthermore, cryotherapy does not lay open lymph and blood spaces and so lead to possible dissemination of malignant cells. This could theoretically happen in treating squamous celled carcinomata by shaving off the bulk of the tumour with a scalpel, as is practised as a preliminary procedure to radiotherapy in some quarters.

In addition, any pain or discomfort associated with cryotherapy can always be obviated by preliminary medication with APC and the use of local anæsthesia.

Finally, there is just one other point I would like to make in connexion with rodent ulcers. It is well known that these carcinomata quite commonly occur at the sites of skin injuries and are not infrequently seen at the excision sites of previous rodent ulcers. The excision operation constitutes a bigger skin injury than did the original "scratch". Patients complain that the first lesion had not been adequately excised. In most cases (knowing who did the excision), the dermatologist is in the sound position of being able to quickly disillusion the patient on that point. The so-called "recurrence" is a new lesion altogether in a person who has a susceptibility to develop epidermal carcinomata as the result of injuries to his or her skin.

Yours, etc.,

J. M. O'DONNELL, M.B., Ch.M.,
Honorary Consulting Dermatologist, Royal Perth Hospital; Honorary Dermatologist, Princess Margaret Hospital, Perth.
Padbury House,
170 St. George's Terrace,
Perth,
Western Australia.

November 28, 1957.

EYE INJURIES IN INDUSTRY.

SIR: May I commend Dr. Lennox Price for his excellent article on "Eye Injuries in Industry" under the heading of "Brush Up Your Medicine" (M. J. AUSTRALIA, November 23, 1957). I hope it will be read and absorbed by many general practitioners who treat such cases. There are some points which can be instructive also to ophthalmologists who are not directly associated with large numbers of eye injuries in an industrial area, especially when compensation courts have to be contemplated. Dr. Price has rightly stressed the importance of taking a careful history of the injury—also a note of the visual acuity in each eye—neglected more often than many realize, and very embarrassing if one is examined in the witness box. There seems to be a tendency of late to discard the pad and bandage after removal of foreign bodies and stain embedded in the cornea. The use of antibiotics has certainly relieved us of many of the infections formerly seen in these cases—but the judicial use of a firm pad and bandage should not be forgotten.

Already this article by Dr. Price has produced results—one large industrial unit in this area has requested a copy for instructions to its ambulance officers in treating eye injuries, and I feel sure the end result will be beneficial to all concerned.

Yours, etc.,

HUGH G. ALLEN.
A. & N.Z. Bank Chambers,
Bolton Street,
Newcastle,
New South Wales.

November 29, 1957.

A CLUMSY KEY TO THE TREASURE OF FREE GOLD.

SIR: As the Free Pharmaceutical List of the Commonwealth Government is due for its quarterly revision, may I point out to its compilers that some of us are confused by (i) gold salts (eighteen preparations in Items No. 11, 14 and 140), (ii) prophenpyridamine para aminosalicylate, (iii) misnomers for (*a*) phenidione and (*b*) aminophylline, and (iv) the omission of Balm of Gilead from the extra-pharmacopœial addendum. I protest about this, because the compilers allow "ointment of galls with opium", "tincture of myrrh" and "ichthammol". Moreover, is it too much to ask of the Public Service that our *vade mecum* be presented in logical format with discernibly rational arrangement and cut the cackle of proprietary advertisement?

Yours, etc.,

GODFREY HARRIS.
87 Macquarie Street,
Sydney,

November 29, 1957.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

PROGRAMME OF COURSES FOR 1958.

THE Post-Graduate Committee in Medicine in the University of Sydney announces the following programme of courses and training for the year 1958. Dates of courses are all subject to confirmation.

Metropolitan Courses.

The programme of metropolitan courses is as follows: March 15 and 16, week-end course in neurology; March 22 and 23, week-end course in electrocardiography; April 12 and 13, week-end course in radiological diagnosis and technique; April 19 and 20, week-end course in occupational medicine; May 5 to 16, general revision course; June 21 and 22, week-end course in gastro-intestinal diseases; July, revision course at the Rachel Forster Hospital for Women and Children; July, course in cardiology in conjunction with Royal Prince Alfred Hospital; July 26 and 27, week-end course in radio-isotopes; August 2 and 3, week-end course in pædiatrics; September 1 to 5, course in gynæcology and obstetrics at the Women's Hospital, Crown Street; October 11 and 12, week-end course in rheumatic diseases; October 25 and 26, week-end course in chest diseases; November 1 to 7, general revision course, Auckland, New Zealand (see separate announcement); November 8 and 9, week-end course in dermatology; November 22 and 23, week-end course in psychosomatic medicine; November 29 and 30, week-end course in mental deficiency, Newcastle.

It is possible that a course in orthopædic surgery will be included in the foregoing programme. Two courses in anæsthesia are also likely to be held.

General Revision Course, Auckland, New Zealand.

Discussions are taking place with the Auckland Medical Post-Graduate Committee for the holding of a week's course in Auckland from Monday, November 3, to Friday, November 7, 1958. These dates have been chosen because it will be possible for those attending the course to leave Australia on Wednesday, October 29, and return on Sunday, November 16, 1958, making a total of 18 days, during which time they can attend the course, and plan a short period of sight-seeing in New Zealand before returning to Australia. Further details will be announced later.

If the course is held, attendance fees and travelling expenses incurred in attending the course will be allowable as income tax deductions.

Resident and External Training Facilities.

Anæsthesia.—Post-graduate residencies are available for practical training in modern methods of anæsthesia at The Royal Newcastle Hospital. Fees, including board and residence, are £5 15s. 6d. per week of five days, Monday to Friday. Post-graduate residencies for full-time residential training in anæsthesia are also available at the Lewisham Hospital for a minimum period of four weeks during the months of October, November and December each year. Fees, including board and residence, are £6 6s. per week. It is likely that two full-time non-resident courses in anæsthesia may be conducted during 1958 by the Department of Anæsthetics and Resuscitation, Sydney Hospital.

Blood Grouping and Transfusion.—On application to the Committee, instruction in the technique of blood grouping and transfusion can be arranged, free of charge, at the Red Cross Blood Transfusion Service or at The Royal Newcastle Hospital.

Gynæcology and Obstetrics.—Post-graduate residencies are available in gynæcology and obstetrics at The Women's Hospital, Crown Street, Sydney, and at the Royal Hospital for Women, Paddington. Fees, including board and residence, are £5 15s. 6d. per week of seven days, and early enrolment with the Committee is essential.

External Studies.—Arrangements can be made with due notice to meet the individual needs of medical practitioners requiring *"à la carte"* courses. Fees are £1 1s. enrolment and £1 1s. per week.

General Residencies.—Post-graduate residencies are available for women graduates at the Rachel Forster Hospital for Women and Children, Redfern. Fees, including board and residence, are £6 6s. per week of seven days.

Post-Mortem Examinations.—On application to the Committee, instruction in the technique of the performance of post-mortem examinations can be arranged free of charge.

Week-End Conferences, Country Centres.

The following week-end conferences in country centres (all subject to confirmation) have been tentatively arranged: March 29 and 30, Tamworth; April 26 and 27, Bathurst; April 26 and 27, Wagga Wagga; April 26 and 27, Newcastle (first); June 14 and 15, Broken Hill; June 28 and 29, Katoomba; July 5 and 6, Hornsby; July 19 and 20, Wollongong; August 16 and 17, Newcastle (second); September 6 and 7, Albury; September 13 and 14, Taree; October 18 and 19, Parramatta; October 22 and 23, Newcastle (third); November 15 and 16, Bega-Cooma.

Annual Subscription Course.

The annual subscription course covers attendance at lectures by overseas lecturers and other specially arranged activities. The annual fee is £2 2s. from July 1. The fee for resident medical officers is £1 1s. A detailed diary card is printed at regular intervals throughout the year and posted to members. Occasionally last-minute alterations to meetings are necessary, and these are notified by advertisement in *The Sydney Morning Herald* ("Public Notices"), if possible on the day before the meeting.

Diploma Courses.

The following information relates to the diploma courses: January 13, M.R.A.C.P. course begins (11 weeks); March 17, the final F.R.A.C.S. course begins (6 weeks) (held in conjunction with the New South Wales State Committee, Royal Australasian College of Surgeons), the D.D.R. I course begins (4 months) and the D.T.R. I course begins (4 months); April 8, the primary F.R.A.C.S. course begins (20 weeks); May 12, the D.D.M. I course begins (12 weeks); June 2, the M.R.A.C.P. course begins (11 weeks); June 9, the D.P.M. I course begins (8 months); July 4, the D.A. II course begins (3 months), the D.C.P. Group I course begins (7 months) and the D.C.P. Group III course begins (7 months); July 21, the D.P.M. II course begins (7 months), the D.D.R. II course begins (8 months) and the D.T.R. II course begins (8 months); August 11, the D.D.M. II course begins (7 months); September 1, the D.A. I course begins (11 weeks), the D.G.O. I course begins (11 weeks), the D.L.O. I course begins (11 weeks) and the D.O. I course begins (11 weeks); September 23, the D.C.P. Group II course begins (6 months); November 17, the D.G.O. II course begins (3 months), the D.L.O. II course begins (3 months) and the D.O. II course begins (3 months).

Method of Enrolment and General Information.

Further information relating to fees and other matters may be obtained on application to the Course Secretary, The Post-Graduate Committee in Medicine in the University of Sydney, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney."

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

SUMMARY OF COURSES TO BE CONDUCTED IN MELBOURNE IN 1958.

THE following schedule has been drawn up as a guide for those who may be planning post-graduate study in Melbourne in 1958.

Courses for Higher Qualifications.

Part I.

Courses suitable for candidates for Part I of the M.D., M.S., M.G.O., D.G.O., D.O., D.L.O., D.P.M., D.D.R., D.T.R., D.A., Primary F.R.A.C.S., Primary F.F.A.R.A.C.S. and Part I of the Diplomas of the College of Radiologists will be held in the following subjects:

Anatomy. The course commences on February 24, and is conducted on Monday and Wednesday afternoons for five months.

Physiology. The course is arranged similarly to that in anatomy.

Pathology. The course commences on March 3, and is conducted on Monday and Wednesday afternoons for four months.

Physics. The course commences on April 10, and is conducted on Thursday afternoons for three or four months.

Microbiology. The course commences on April 15, and is conducted on Tuesday afternoons for 15 weeks.

Psychology I. The course commences in mid-March, and is conducted for five hours per week till late October.

Part II.

The following courses are for Part II candidates:

A course in medicine, suitable for candidates for senior medical qualifications such as M.D. or M.R.A.C.P., will commence on May 26, and continue each afternoon till July 19, at Prince Henry's Hospital.

A course in surgery, suitable for candidates for senior surgical qualifications, such as M.S. or F.R.A.C.S., will commence on March 17 and continue in the afternoons till April 24, at the Royal Melbourne Hospital.

A basic course for the diploma in microbiology will commence on April 15 and continue on Tuesday afternoons for 15 weeks. A course in basic pathology will commence on March 3 and continue on Monday and Wednesday afternoons for four months.

A course in radiodiagnosis for the D.D.R. and Part II of the D.C.R.A. will commence in May and continue part time for about ten weeks; a course in special pathology will commence in July and continue part time for six weeks.

Courses in radiotherapy and special pathology for the D.T.R. and Part II of the D.C.R.A. will be arranged when candidates present.

A course in psychiatry for the D.P.M. will commence in mid-May and continue part time for three months. There will be a course of 15 lecture-demonstrations in neuropathology, commencing in June, and a course in psychopathology will commence in March and continue part time for eight months.

A course in laryngology, otology and pathology for the D.L.O. will commence in the first week in June, if sufficient candidates present, and continue part time for three or four months.

Courses in ophthalmology and special pathology for the D.O. will commence on April 14 and continue part time for four or five months.

Courses in gynæcology, obstetrics and special pathology for the M.G.O. and the D.G.O. will be arranged when sufficient candidates present.

A course in the theory and practice of anæsthetics for the D.A. and the F.F.A.R.A.C.S., consisting of three lectures per day, will commence on March 17 and continue for two weeks.

Refresher Courses.

A gynæcology and obstetrics refresher course for recent graduates will commence at the Royal Women's Hospital on February 10, and continue full time for two weeks. Commencement of the course depends on the receipt of sufficient enrolments.

A medical and surgical course for general practitioners will commence on April 28, and be conducted at metropolitan hospitals for one week, full time.

A gynæcology and obstetrics refresher course for general practitioners will be conducted at the Royal Women's Hospital, full time, for two weeks, commencing on August 11.

A post-graduate week will be conducted at the Royal Children's Hospital on a date to be determined.

A psychiatry course will commence in March and continue part time for three months.

Conduct of Courses.

Courses in medicine and surgery, as set out above, will be conducted by the honorary staffs of Prince Henry's and the Royal Melbourne Hospitals respectively. The Ophthalmological Society will conduct the ophthalmology and special pathology course, the Victorian Division of the Australian

Oto-Laryngological Society the course for D.L.O. II. The Australian Association of Psychiatrists will conduct the course in psychiatry, and the Mental Hygiene Authority the course in neuropathology, both for the diploma. The University of Melbourne will conduct the courses in psychology and psychopathology. The Faculty of Anæsthetists will conduct the course in theory and practice of anæsthetics, and the Mental Hygiene Authority the psychiatry refresher course. All other courses shown will be conducted by the Melbourne Medical Post-Graduate Committee.

Enrolments and Inquiries.

Enrolments for all courses should be made through the Melbourne Medical Post-Graduate Committee, except for the courses in psychology I and psychopathology, the psychiatry refresher course and the course in anæsthetics, for which they are made with the body conducting each, as outlined above. The Director of the Post-Graduate Committee will be pleased to answer inquiries concerning the 1958 programme. Correspondence should be addressed to him at the office of the Melbourne Medical Post-Graduate Committee, 394 Albert Street, East Melbourne, C.2, Victoria.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *etc.* are promulgated in the *Commonwealth of Australia Gazette*, No. 61, of November 7, 1957.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Confirmation in Rank.—Surgeon Lieutenant (for Short Service) (on probation) Richard George Hingston is confirmed in the rank of Surgeon Lieutenant (for Short Service), with seniority in rank of 24th August, 1956.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Flight Lieutenant Robert John Limbury Tebbutt (0211575) is transferred from the Reserve and appointed to a short-service commission, on probation for a period of twelve months, 27th May, 1957.

Alex Kauler (055421) is appointed to a permanent commission, on probation for a period of twelve months, 15th July, 1957, with the rank of Pilot Officer.

The following are appointed to a temporary commission with the rank of Pilot Officer (Student): David Walters (0242918), 31st January, 1957; Ronald Philip Quirk (0314324), 8th May, 1957.

The probationary appointment of the following Flight Lieutenants is confirmed: W. J. Bishop (0210441), J. N. Burry (0310758).

Flight Lieutenant (Acting Squadron Leader) R. P. V. Checcucci (0311611) is granted seniority in the rank of Flight Lieutenant as from 11th July, 1955.

The following Flight Lieutenants are granted the acting rank of Squadron Leader: E. H. Stephenson (0312182), 7th June, 1957, W. J. Bishop (0310441), 1st July, 1957.

The resignation of the following Flight Lieutenants is accepted: R. H. Mackay (036622), 28th June, 1957; T. J. Orr (024805), 5th July, 1957; E. Solo (014782), C. F. Campbell, D.F.C. (014785), 9th August, 1957.

The Royal Australasian College of Physicians.

SCHOLARSHIPS AND RESEARCH GRANTS.

THE Council of The Royal Australasian College of Physicians announces the following awards of scholarships for 1958: Travelling Scholarship in Medicine, Dr. H. Lander (South Australia); Wunderly Travelling Scholarship in Thoracic Disease, Dr. John Read (New South Wales).

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 23, 1957.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3(3)	2(2)	2(1)	..	..	..	..	..	7
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	10	..	..	..	..	..	10
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1	1	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	7(2)	7(7)	..	..	2	..	5	..	21
Diphtheria	..	1(1)	..	..	..	..	..	..	1
Dysentery (Bacillary)	..	..	2(2)	..	2(1)	..	2	..	6
Encephalitis	..	1(1)	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	51(28)	18(10)	8	5(3)	9(3)	..	..	..	91
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	3	..	3
Leptospirosis	..	..	2	..	..	..	..	..	2
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	2	4(4)	1	1(1)	..	..	..	..	8
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1	..	..	..	..	..	..	..	1
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	74(65)	17(17)	33(17)	40(35)	..	1	2	167
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	11(5)	10(9)	1	5(2)	..	..	..	..	27
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	1(1)	..	1	..	2
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	40(20)	13(11)	17(5)	4(3)	6(5)	..	5	..	85
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1(1)	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

The following awards have been made from the Adolph Basser Research Fund:

Dr. R. A. Joske, of Melbourne: a Fellowship for a period of five years for research into gastro-intestinal disease and chronic liver disease, in the Department of Medicine at the University of Western Australia.

Dr. Denis F. J. Halmagyi, formerly of Hungary: a Fellowship for a period of five years for research into the pulmonary circulation, in the Department of Medicine at the University of Sydney.

Dr. K. D. G. Edwards, of Sydney: a Fellowship for one year for research into body fluid, electrolyte and renal function, in the Clinical Research Department of Sydney Hospital.

Dr. B. P. Billington, of Sydney: a grant-in-aid for part-time research into blood groups and disease, in the Department of Medicine at the University of Sydney.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1957:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1*a*) of the Act: Bennett, Donald Collier, M.B., B.S., 1946 (Univ. Adelaide); Bradfield, Geoffrey Frank, M.B., B.S., 1952 (Univ. Queensland); Glass, Reuben David, M.B., B.S., 1955 (Univ. Melbourne); Moore, Francis Morrissey, M.B., B.S., 1940 (Univ. Melbourne); Oxbrow, Douglas Wade, M.B., B.S., 1953 (Univ. Melbourne); Powrie, Robert Malcolm, M.B., B.S., 1956 (Univ. Adelaide), B.Sc., 1949 (Univ. Western Australia).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1*b*) of the Act: Bellamy, John Merlin, M.R.C.S. (England), L.R.C.P. (London), 1937, B.A., 1932 (Univ. Cambridge); Brennan, John Dermod, M.B., B.S., 1955 (Univ. London); Davis, Raymond James, M.B., B.S., 1949 (Univ. London); Gill, Paul Michael, M.B., Ch.B., 1956 (Univ. Bristol); Helme, Alan Crichton, M.R.C.S. (England), L.R.C.P. (London), 1952; McNeill, Roger, L.R.C.P. (Edinburgh), L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1949; Noakes, Norma Helen Wickham, M.B., B.S., 1953 (Univ. London), M.R.C.S. (England), L.R.C.P. (London), 1953; O'Sullivan Beare, Donal Barry, M.B., B.Ch., 1927 (Univ. Dublin).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1*c*) of the Act: Soller, Karla, M.D., 1945 (Univ. Prague).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (2*a*) of the Act: Rado, Tibor, M.D., 1918 (Univ. Budapest).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (2*b*) of the Act: Raneri, Pietro, M.D., 1948 (Univ. Messina).

Registered medical practitioners who have been issued with licences under Section 21*c* of the Act: Luedecke, Hans, M.D., 1946 (Univ. Munster); Malesevic, Bosko, M.D., 1924 (Univ. Graz); Peukert, Joseph, M.B., 1944 (Univ. Prague); Staszkiw, Wladimir, M.D., 1936 (Univ. Lwow); Todorovic, Michailo, M.D., 1936 (Univ. Belgrade).

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Kennedy, Michael Andrew, M.B., B.S., 1956 (Univ. Sydney), 70 Roslyn Gardens, Elizabeth Bay, New South Wales.

O'Higgins, James Patrick Dermott, L.A.H. (Dublin), 1949, 87 Dalhousie Street, Haberfield, New South Wales.

Deaths.

THE following deaths have been announced:

LEITCH.—Emily Bertha Leitch, on November 10, 1957, at Melbourne.

KELLY.—John Horace Kelly, on November 26, 1957, at Toorak, Victoria.

GOLDMAN.—Norman Goldman, on November 30, 1957, in an accident near Tamworth, New South Wales.

Diary for the Month.

DEC. 17.—New South Wales Branch, B.M.A.: Medical Politics and Ethics Committees.
DEC. 27.—Queensland Branch, B.M.A.: Council Meeting.
1958.
JAN. 6.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 7.—New South Wales Branch, B.M.A.: Council Meeting.
JAN. 10.—Tasmanian Branch, B.M.A.: Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

Queensland Branch (Honorary Secretary, 88 L'Estrange Terrace, Kelvin Grove, Brisbane, W.1): All applicants for Queensland State Government Insurance Office positions are advised to communicate with the Honorary Secretary of the Branch before accepting posts.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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